MAY, 1942

Orthodontics

Office of the Editor: 8022 Forsythe Boulevard, St. Louis
Published by The C. V. Mosby Company, St. Louis

American
Journal of Orthodontics
and Oral Surgery

VOL. 28 MAY, 1942 No. 5

Original Articles

HUMAN TISSUE RESPONSE TO ORTHODONTIC INTERVENTION OF SHORT AND LONG DURATION

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THE problems of tissue changes following orthodontic measures may seemingly be considered already solved by the experimental work that was started as far back as 1905 by Sandstedt;[51] still many authors, inspired by this work, continued research in this field. Many of their findings are listed in the bibliography. Many questions, however, remain unsolved, and, as research continues, additional facts are being revealed. This paper is intended to widen our knowledge of the susceptibility of the human dental tissues (first recognized by the author in 1936[39]) and to produce further evidence of the great specific vulnerability of the dental cementum.

The fact emphasized by the author in previous publications,[38, 39] that anything like physiologic tooth movement is impossible with the means at our disposal, is to receive still further proof in this study. Since the infliction of damage cannot be avoided, as these new findings definitely show, the operator is again admonished to cause the least amount of injury possible by using the lightest force at long intervals. Nature should be given enough opportunity and time to perform what we intend to do by the application of stimuli, not forces. Stimuli alone create the osteoclasts, which remove the obstacles to tooth movements. They, not the appliances, are responsible for our results. Appliances are only a means to create osteoclasts by stimulation. The necessity of the use of light forces, announced as far back as 1911 by the author,[37] is accepted now by most of the leading men in our profession. Orban,[44] for instance, accepts this standpoint in saying, "Every orthodontic moving of a tooth by means of appliances is an overstress in the biologic sense." This opinion is emphasized

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by others like Skillen and Stuteville. In his experiments on dogs, Skillen[57] came to the conclusion "that a greater amount of rotation was obtained in five days with a lesser amount of force than was obtained in three days with a greater force."

When Stuteville,[61] in number 9 of his conclusions, says, "there should enough time elapse between adjustments to allow all the supporting tissues to return to normal," he seems to agree in this respect. Continuous forces—light though they may be—are always too strong, because the tooth movement is quicker than the time required for bone resorption. The forces can never be *too* light. With light forces, the damage is never as great as when strong forces are used. When we treat with light pressures, our only possible loss is time. So far, however, we must admit that we have no knowledge regarding the ultimate effect of light forces used over a long period. The author has been able to find only one reference to the effect of intermittent forces used over a longer period of time in man. Hemley,[21] after the use of a bite plate for eleven weeks, states that the histologic sections of the mandibular incisors "did not show evidence of bone destruction or of cementum destruction."

We have a considerable amount of proof that, in the use of strong forces, the tissues of the periodontal membrane are crushed; bone, the obstacle to the tooth movement, can now only be removed by resorption of undermining nature, viz., through osteoclastic activity in nearby areas. Orban[44] says: "Resorption of bone and clearing away of necrotic tissue fragments can continue for months and considerable time pass before the tissue has so much recovered that it can accomplish the desired resorption." If this happens at the alveolar crest, a great amount of bone substance, so important in this very area, is lost forever. The alveolar bone supporting the root is shortened, and lasting damage may result. Gottlieb and Orban[14] say: "This (viz., disappearance of the bone at the alveolar crest) is an apparently lasting damage and hardly any repair follows." In cases where the injury is lighter, a repair of the alveolar crest may take place, restoring the length of the alveolus to some extent.

The author does not intend to discuss here again the practicability of intermittent forces versus continuous forces. That phase was dealt with at length in earlier papers.[38, 39] In order, however, to justify his unchanged position, i.e., that intermittent pressure is better, he will quote from a few more authors. Pommer,[46] one of the outstanding men in anatomy and pathology, states "that the functional trophic stimuli manifest themselves in intermittent pressure." Zschokke,[69] while saying "continuous and intermittent pressures act quite differently," prefers the latter. In his latest paper, Kaare Reitan[31] advocates "intermittent forces or continuous forces followed by rest periods to avoid root resorptions and to allow the tissues to adjust themselves."

Ziebe[68] found in his experiments on dogs and monkeys "that the movement of the teeth as well as the bone transformation continues for a long time after the force of the appliance had been discontinued." Basing on his clinical experience, Mershon years ago made a similar statement.

These observations, with others mentioned elsewhere,[38, 39] justify the use of intermittent forces, though it must be admitted that histologic findings,

such as reported by Ziebe, have not yet been corroborated in humans. We seem to have proof, however, from practice on the human also, when we observe the results in patients treated with intermittent pressure applied only at night, as witness the head cap. A cementum resorption, once started, may continue even if the orthodontic force is stopped, as has been shown by Orban[45] and Gottlieb and Orban.[14] Skillen and Kriwanek[56] saw bone resorption still active thirty days after removal of bands and ligatures when gum inflammation was caused by them.

Many interesting questions still await clarification. The reaction to the same amount of force varies with each person, depending on still unknown systemic or other conditions. We have also little knowledge of the later and ultimate (20 years later) effects of orthodontic work. We will not arrive at a solution of these problems until closer cooperation has been established between orthodontists and general practitioners, who see our patients many years later, when we have lost track of them. Then, and only then, will we know whether the benefits of our treatment outweigh their harm. From his practical experience as careful an author as Mershon[34] has the courage to say: "Of the cases that we have improved materially we hope the percentage is high, but I am very much afraid the percentage is also high of those in which we have done harm."

Not only the patient's general constitution, but also his condition at the time of treatment is of decisive importance. We have to agree with Winkler:[66] "The readiness for the reaction of the organism certainly is not only different in different individuals, but also in the same individual at different times." It is agreed that this readiness of the cells for reaction is subject to continual change. The fundamental biologic law of Arndt-Schulz states "that light stimuli stir up the vitality of the cells, while medium strong forces encourage, strong forces reduce, and very strong forces eliminate, this vitality. The dosage of the forces to be used cannot be fixed by a simple scheme, as each individual in health or disease reacts differently."

I would like to quote a few authorities on this subject: Orban[43] says, "different individuals react in different ways to the same outer influences," and again,[44] ". . . the resorbability of the cementum surface is certainly different in different individuals. . . ." Gottlieb,[13] ". . . it is well illustrated how the same cause brings about different results on different teeth—even on different places of the same tooth." Skillen:[55] "Why, under apparently similar conditions, root resorption occurs in some instances and not in others is still a problem. . . ." Stillman and McCall[59] emphasize the importance of the individual tissue reaction as proved in different cases of traumatic occlusion, "where in one case a tooth subjected to 'plus-occlusion' may become strengthened and in another case weakened, depending on the ability of the supporting tissues to adjust themselves to altered occlusal conditions."

Kronfeld,[28] "In every tissue reaction the resistance of the cells is of decisive influence, and since the limit of this resistance is unknown, it is impossible to determine how much irritation is necessary to produce a certain form of tissue reaction," and again, "In individuals with high resistance even a great amount of occlusal trauma does not produce resorption of the root. On the

other hand, in patients susceptible to root resorption, normal mastication or careful orthodontic treatment may occasionally produce progressive root resorption leading finally to complete destruction of the root." The same difference in individual reaction, we encounter in the pulp (Kronfeld[27]). (Kronfeld and Schour,[30] "For this phenomenon (the unequal distribution and degree of enamel hypoplasia) we have no explanation beyond the general truism that among all cells of the body, there must be individually different resistance to injuries of any kind.") (In his experimental studies on bone transplantation in rats, Stein[58] came to the conclusion "that the different results in the same operation may be accounted for by the individual differences of the animals," all conditions being equal.)

In one of his articles, giving the general rules for the treatment that must always be kept in mind, Mershon says: "the amount of force is different with every patient, the same as drugs administered have to be different." Brodie,[7] "The fact remains that, regardless of appliances used, forces exerted, or the duration of the treatment, there are wide variations in tissue tolerance or the susceptibility to resorption." (Mershon[35] and Dewey[8] previously pointed out "that biology cannot be worked out by mathematical formulae. . . . You cannot regulate teeth by mathematical rules or calculations.") The author also could prove from his human material that the repair of cementum resorptions shows very great individual differences.[39] While, in some cases, the resorptions were entirely repaired after a relatively short time, the author could see in other cases that, even after a long recovery period, the resorptions were only slightly repaired by secondary cementum.

On these findings, which proved true not only for the cementum but for all tissues involved, the author based the statement that we are groping entirely in the dark. We surely will often be embarrassed, if we rely on the same duration of retention for all patients alike. On a previous occasion[38] the author has dealt with this question of retention using monkeys as experimental material. It is now generally accepted by the profession that the reaction to treatment varies with the individual, though the knowledge of this fact in many instances does not guide the practical procedure.

Stuteville,[60] also, admits this different tissue reaction in individuals, saying: "It is admitted that there are great individual differences in patients. Some respond to orthodontic treatment much more rapidly than others." Yet on another occasion[62] he asserts: "We are able to make predictions as to the tissue changes that will be obtained by a given force." This second statement cannot be accepted. It is not in keeping with the generally accepted views and contradicts his own statement quoted first. Waldo[67] states: ". . . bone is not the same in all individuals," and suggests "that this variation may have an important bearing upon the success and failure of the orthodontic treatment."

Why do I quote these authors on this topic? Because we still find, even in the most recent publications, the routine practice and advice for using indiscriminately certain measured forces to bring about the wished-for changes, and "to be sure," in doing so, one would avoid injuries to the tissues concerned. This kind of procedure is advocated even by men who gave expression to their conviction that the tissue reaction to the same extrinsic influences is

different in each individual. This demand for the use of only measured forces has been accepted without due criticism or control ever since the publication of Schwarz's paper.[54] It is now perpetuated by repetition.

Since we are quite in the dark as to whether an individual has a high tissue resistance, or is very susceptible to damage, I consider it unjustified and wrong to use indiscriminately the same measured amounts of force for all persons, even disregarding their ages. I repeat again: We have only one reliable clinical criterion for the correctness of the forces applied in any given individual, i.e., the firmness and nonsensitiveness of the teeth. The patient should not be forced to alter his diet during treatment, nor avoid normal hard food because of the soreness of his teeth. Looseness in most instances is the clinical sign of some lost bony or ligamentous support, or both. Sensitiveness and soreness are the clinical signs of present or recent inflammation, hemorrhage, or destruction.

There may be only one indication for the use of measured forces: animal experimentation. As the animals cannot tell us whether or not their teeth have become sore, we have to use some guide as to the amount of force applied. But it holds true also here that the various tissues react differently.

All the tissue changes observed after orthodontic intervention should not be considered as having been brought about by this intervention alone. Only the comparison of a treated tooth with a control tooth in the same individual may serve as a reliable clue to the conditions found. Even in untreated teeth we occasionally see cementum resorptions (Fischer[11]), which according to Euler and Mayer[9] and others are found often in teeth that have never been treated orthodontically. In contradiction to Fischer and Euler and Mayer, Kronfeld[27] says that "all resorption in teeth is the expression of a pathological process or injury."

Cementum resorptions after application of extrinsic artificial forces by which the pressure in the periodontal membrane is increased above the normal are unavoidable in man, and therefore are always present, as is proved by the evidence submitted by the author on previous occasions.[39a, 40] The cementum scars never disappear and give us reliable information as to the past of the tooth. This fact is now generally accepted, and even Stuteville,[61] who at first contradicted me, comes finally to the conclusion that, "it is safe to say that root resorption* is produced in all cases of malocclusion which are corrected by orthodontic appliances." Skillen[57] says, on the basis of his experimental work, that even in dogs whose cementum is supposed to be, and really is, much more resistant than that of man, "tooth resorption near the area of greatest pressure . . . was of common occurrence." Similar findings were reported by Kaare Reitan[51] in his experiments on dogs.

The author will prove in this paper that Stuteville[61] and other authors (Gottlieb-Orban[14]) are altogether wrong in overemphasizing that "the human cementum . . . reacts less readily than bone to pressure by resorption."

*I would suggest that the terms root resorption and cementum resorption be not used interchangeably. While root resorption denotes a real shortening of the root, the cause of which is not yet known, cementum resorption means only a loss of tooth substance not necessarily localized at the apex. The cause for its occurrence is well known. While root resorptions occur relatively seldom, cementum resorptions are always present in orthodontic procedures.

Pathologic or involutionary conditions are found not only in the cementum of untreated teeth of normal individuals, but such regressive and pathologic changes may also be encountered in the pulps of untreated teeth and even in the pulps of tooth germs, where extrinsic causes cannot be held responsible. Partial disintegration of the odontoblasts, edema, vacuole and cyst formation are sometimes found. Here also, in evaluating the effects of orthodontic interference, comparison with untreated teeth of the same individual will be a sure guide in our judgment and interpretation of findings. Mineral metabolism, infectious diseases (influenza) (Weinman[64]) and local conditions (crowding) have a decisive influence on the normality of the tissues of the paradontium. Perhaps the vulnerability of the alveolar process and the other tissues of the paradontium can be accounted for by the fact that they, according to Thoma,[63] have to be considered as end organs with quite an unstable equilibrium.

Though control teeth were most desirable, they were not obtainable in some of the cases to be shown here; but the damages were so pronounced and severe that they can only be considered as having been caused by the forces applied which heretofore nobody (myself included) thought capable of inflicting great harm. My statement that the human cementum is a "most vulnerable tissue" will be corroborated by my new findings. This statement was not accepted by Orban:[44] "I cannot let pass the assertion of Oppenheim 'that the cementum is the most vulnerable tissue of the periodontium,' and we have always thought the contrary to be true."

Neither did Orban share my doubts that, from animal material, conclusive inferences can be drawn as to conditions in man. Orban in collaboration with Gottlieb, mostly on dogs,[14] made categorical deductions as to conditions in man, though on a previous occasion, Orban[41] made a statement that would suggest that he too was of the opinion that exact assumptions from dog to man cannot be drawn "on account of the great differences in the normal anatomic conditions." I have an advocate of my opinion, among others, in Kronfeld[28] who states ". . . because of the possible differences in tissue reaction between man and animals, the results obtained in animals are of limited value." Moreover the forces used by Gottlieb-Orban[14] and by Orban[44] in their dog and human material were not of an orthodontic nature. Orban[44] states "that excessive irregular stress was working on the teeth." But we need to know the tissue reaction to light or strong orthodontic forces, not to excessive ones. Excessive forces have no place in orthodontics.

In the literature we oftentimes find optimistic statements about the ultimate fate of our treated cases, which are not justified when we face the fact of unavoidable damage; since the latter is a fact that cannot be denied, optimistic assumptions are not warranted, for they are not proved.

In dealing with the question of apical root resorption, Brodie[7] makes the statement: ". . . it should not cause such grave concern as it has occasioned in the past. We do not condemn surgery for leaving scars—we accept them as a price that must be paid for a benefit."

I doubt whether some changes shown in a previous communication[39] or in specimens VIII and IX of this publication or root resorptions like those shown by Ketcham[24, 24a] or lately by Hemley,[22] can be looked upon as being of benefit.

Though Rudolf[50a] reports that in 439 orthodontically treated cases, apical root resorption was found in 69.24 per cent (which really should cause alarm), he comes to the conclusion that, "it is possible that the foreshortening of a few roots . . . might not have been too large a price to pay for other advantages gained by treatment."

Stuteville[60] makes the broad statement: "from these experiments it is evident that the much-feared injuries supposed to be caused by orthodontic appliances are more theoretical than real." The evidence given below is proof enough that the injuries are not merely theoretical, but *real* injuries.

The quoted opinion of these writers may be looked upon as representing the general attitude of the profession toward the ultimate fate of orthodontically treated cases. It is really a very optimistic one, though the author himself cannot give a contradictory report as our knowledge of the ultimate fate of our cases is still very limited.

On the basis of the new findings presented in this paper, a note of warning should be repeated. In regard to the frequent occurrence of apical root resorption, an optimistic attitude is not warranted, because shortened roots never can resist the stress of function as well or as long as teeth with roots of normal length. This situation becomes worse with advancing age as the extra-alveolar lever with all its accompanying disadvantages always becomes longer through the normal involutionary process.

"Under physiologic conditions no resorption occurs" (Kronfeld[28]). This is as generally accepted now by the profession as the fact that in artificially moved teeth cementum resorptions cannot be avoided. But the potential severeness and danger of the injuries are not yet realized, though some evidence on this subject has already been published. Still more evidence will be submitted here.

Basing on their experimental dog material, Gottlieb-Orban[14] stated that, "every resorption may be considered as dangerous for the tooth," and, "resorptions on the whole are incalculable." Orban[45] says: "Sometimes resorptions are not repaired but may continue until the tooth becomes completely detached and lost." Gottlieb-Orban[14] have shown in monkey teeth that, even after a retention period of sixteen days, many osteoclasts were still at work in the depths of the excavated areas.

One might assume that on account of the loss of cementum and bone substance and the, therefore, wider periodontal space, the stimulus of pressure was no longer present, and that the osteoclasts had disappeared. What may be therefore of advantage sometimes, if it happens in the bone, may be disadvantageous if it happens in the cementum; but we have no means of inducing the one or of preventing the other. The damages, if we disregard the time factor, depend mostly upon the amount of force as will be shown in this treatise. The warning, therefore, should again be repeated: use light forces, and when encountering great resistance, do not double your forces; double your time! Do not try to establish speed records. The experience that light forces work more efficiently than strong ones was noted not only by Skillen[57] but also by Ziebe[68] in his experimental work on dogs and monkeys where he proved that this rule

applies also to traction. In widening the upper jaw, he found "that after using light forces for a long time, the borders of the median suture showed a great amount of bone apposition while, in the use of strong or very strong forces, the limit of the tensibility of the fibers was surpassed; the damages brought about were so severe that the bone response failed to occur, thus prolonging the time of treatment."

The material investigated until now by different authors has consisted mostly of teeth that were moved in various ways for hours, days, weeks, or quite a few months. The author will show here what happens after one day and after several years of treatment. The material was obtained partly by the author, partly by other orthodontists in this country.

The latter furnished the teeth without adherent bone; these teeth had to be extracted for various reasons after different periods of treatment. Only a small part of the material at the disposal of the author will be presented in this paper. The orthodontists from whom I received the specimens are not beginners but quite expert workers. We may, therefore, assume that the findings are not unusual, or brought about by incompetence, but might be expected to be still worse in the hands of less experienced men who change, or are forced to change, their plan of procedure during treatment and shift the teeth first in one direction, then another. The first two specimens to be shown—besides a normal tooth—reveal the tissue reaction after the application of force for one and four days, respectively. The next four specimens deal with the changes caused by silk and wire separation, respectively, after twenty-four hours. The separation was performed in the routine way in order to secure space for making and putting bands on the teeth. The last two specimens reveal in one case the changes produced after three years of treatment with lingual arch and intermaxillary elastics, and in the other the changes observed after one and one-half years' treatment with an edgewise arch.

Experimental work on a larger scale with regard to the changes caused by separation of the teeth was performed by Rebel and Mayer[50] in 1934, though Gottlieb-Orban[14] previously referred to similar experiments in 1931, but these later experiments on monkeys lasted four and five days.

Separation is a common procedure in orthodontics, and was and is recommended by all the leading men and teachers in our profession, as a necessary means of obtaining room for fitting bands. Most of these authors mentioned the sensitiveness of the teeth after such a procedure, but none of them even suspected the possibility of injuries that may occur.

Rebel-Mayer,[50] who conducted their experiments on dogs and monkeys, came to the conclusion that a transformation of all the tissues concerned, hard and soft, has to occur, regardless of whether the separation is of short or long duration. After separation with an ivory separator for one and one-half hours and three hours, respectively, they state, "the effect was extraordinary," and after three hours' separation, "the resorption of the alveolar wall had started already," and, "frequently crushing and tearing of the tissues were encountered." After from three to four hours of separation with rubber, they found crushing of the soft tissues, "a larger resorption in the bone," and "the beginning of a biologic reaction." After separation for three hours with a

wooden wedge, "little bone resorption" was found. No resorptions in the cementum are mentioned. After three days of separation with rubber, they found pronounced tipping of the tooth with deep resorptions at the roots and striking bone formation on the traction side. By this tipping of the apex moreover "the tooth germ of the neighboring tooth . . . showed a shallow but somewhat extended, quite fresh, resorption"; "the whole alveolar wall is in a state of transformation." All this proves that the damages inflicted are in correlation with the amount, rapidity, and duration of the applied force.

A biologic reaction of the bone in dogs was found by Gottlieb-Orban[14] as early as twelve hours after application of force, "along the whole alveolar wall resorptions are found. . . ," while the cementum even after three days of force application in the same dog remained uninjured. We will see what happens in the human cementum after silk and wire separation of only one day's duration.

Before we proceed to the description of our specimens, parts of a normal tooth (Specimen I) may be shown; it is an untreated tooth from the same patient as Specimens II and III. All these teeth were removed under identical conditions. The difference in the findings, therefore, can be attributed to the application of force and not to the trauma of the extraction or some post-mortem changes of shrinkage, incomplete fixation, or other accidents as was suggested by Stuteville when referring to some findings in my specimens.

Fig. 1, Specimen No. I.—Buccal alveolar plate in high magnification; *P*, periodontal membrane; *c*, tissue debris; *O*, osteoid bone; *Ob*, osteoblasts.

Specimen I (No. 17), normal, 12 years of age, maxillary premolar. Some bone of the buccal alveolar crest that was removed with the tooth is reproduced in higher magnification in Fig. 1; the tissue (*c*) crushed during the extraction lies between the root (supposed to be on the left) and the periodontal membrane (*p*) that became detached from the tooth during the extraction.

The bone surface facing the tooth and the alveolar crest is smooth and covered with a thin layer of osteoid bone (*o*) covered with a regular row of osteoblasts (*ob*). It may be admitted that this is a somewhat rare condition found, for we know that bone is constantly undergoing a process of tearing down and rebuilding, some osteoclasts always being found between the bone builders. A part of the pulp reproduced in Fig. 2 shows the regular arrangement of the odontoblasts (*od*) and several vessels in cross and longitudinal sections (*a* and *b*). For special reasons to be given later, I should like to point out that in these vessels no blood corpuscles are to be seen. We see only the cells characteristic for vessels, i.e., the endothelial cells. In the longitudinal sections (*b*), the cells that can be seen are also endothelial cells, not blood corpuscles.

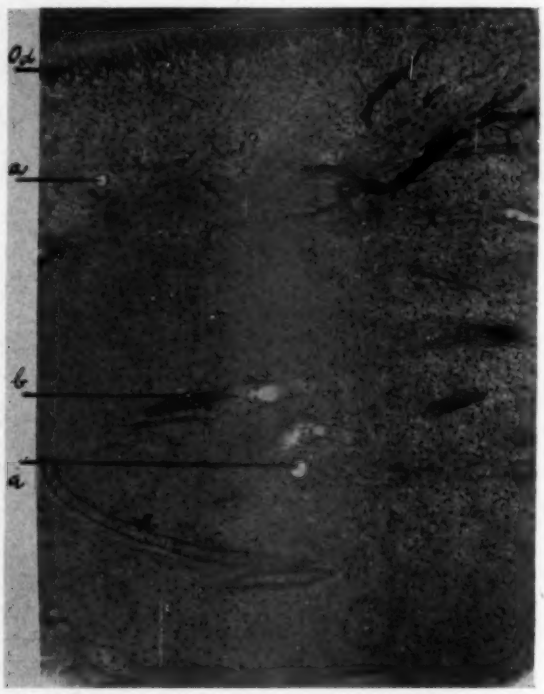


Fig. 2, Specimen No. I.—Normal pulp; *Od*, odontoblasts; *a*, *b*, vessels.

Specimen II (No. 69). This tooth is a mandibular premolar from the same patient as Specimen I. It was moved buccally for twenty-four hours by a spring of stainless steel 0.2 mm. thick, 1½ cm. long, and soldered to a lingual arch.

Fig. 3 is the outline picture and shows the still open foramen and some bone (*B*) on the buccal side that was removed with the tooth. The grooves on the right side of the root surface (Fig. 3*A*) below the bone are artifacts. In a higher magnification of the alveolar crest, we find it surrounded by osteoid bone (Fig. 4*O*), while the surface facing the root shows the distinct signs of resorption. It is scalloped, although in the Howship lacunae there are only scanty multinucleated osteoclasts (Fig. 4*Oc*). The periodontal membrane in this section (Fig. 4*p*) does not reveal any remarkable changes. In following the series, the bone surface below the alveolar crest and just above the artifact (Fig. 5*A*) is smooth again (Fig. 5*Ob*), but in the periodontal membrane we find

the beginnings of a formation already described in a previous communication[39] as a cushion of vessels (Fig. 5*V*) formed and fully restricted to the pressure side in the different specimens. It was and is again interpreted as a device of nature for diminishing or dissipating the applied pressure, so as to act as a shock absorber. This formation is reproduced in high magnification in Fig. 6 where the endothelial cells on each of the capillaries are distinctly seen (Fig. 6*Ec*). Compare these endothelial cells with those in the pulp vessels (Fig. 4*OEc*). In most of the capillaries like those shown in the normal pulp (Fig. 2*a*, *b*), no blood corpuscles are present, though in some of them they are present (Fig. 6*Bc*).

Fig. 3.

Fig. 4.

Fig. 3, Specimen No. II.—Outline picture; *B*, buccal alveolar crest; *A*, artifact.

Fig. 4, Specimen No. II.—Alveolar crest of Fig. 3 in high magnification; *P*, periodontal membrane; *O*, osteoid bone; *Oc*, osteoclasts on the inner scalloped surface of the bone; *C*, cementum.

The periodontal membrane is of the highest importance for us and deserves the greatest consideration, for in all our tooth movements, it is this membrane with which we have to deal first, and the outcome of all our endeavors depends entirely on its readiness for reaction and the consequences of this reaction, the resorption and apposition of bone. The inevitable changes in the other tissues, to be sure, cannot be overlooked but may be classified as accompanying occurrences. The capillaries found in the periodontal membrane consist often only of endothelium like the venules of the thyroid, and are often devoid of blood corpuscles in the same manner as the small arteries in general (Schaffer[52]). Such an accumulation of small vessels as shown in Fig. 5 and former specimens has been observed by previous authors, and its presence was also interpreted as a means to overcome undue stress. Dealing with the description of the periodontium in rabbits, Orban[42] says: "In the periodontium, many wide capil-

laries are to be seen. . . . One may assume that these vessels not only serve the nourishment of the tissue but that they form a kind of elastic cushion in order to decrease the pressure of the tooth against the bone." The same formation of vessels was found and described by Bauer[1] in the periodontium between a deciduous tooth and its successor: "Some of the large veins are in immediate contact with the bony spicules . . . and fulfill an osteoclastic activity. Especially remarkable is the richness of enlarged and mostly empty veins. . . ." At another occasion Bauer[2] attributes to this vessel formation the significance of a cushion; he writes: ". . . many vessels in the shape of glomerules and meshes are found in the posterior part of the capsule of the disk (of the temporomandibular articulation) and doubtlessly they serve as a cushion like the vessels in the periodontal membrane described by Wedel, Schweitzer and Häupl, and Lang. . . ."

Fig. 5, Specimen No. II.—Neighboring slide to Fig. 4; *P*, periodontal membrane; *Ob*, osteoid bone on the inner smooth bone surface; *C*, cementum; *V*, cushion of vessels; *M.c*, epithelial rests of Malassez; *A*, artifact.

Kronfeld[29] confirmed similar findings on the pressure side of one of his specimens and interpreted them as a "great number of enlarged blood vessels." This vessel formation has been interpreted, however, by Stuteville[62] as "vacant areas caused by autolysis of the necrotic connective tissue . . . together with tearing during removal followed by shrinkage during preparation; . . . they are blind cavities lined by peridental membrane tissue, which gradually blends in with the surrounding vital tissue at the termination of the tear." I think that my interpretation is right and that these "vacant areas" cannot be looked upon as artifacts. Skillen[55] could not verify the cushion formation: "To date, nothing has been found which appears to correspond to the angioma-like arrangement described by Oppenheim."

It was stated by the author[39] and corroborated by Kronfeld[29] that this formation may be created only under certain conditions. Kronfeld says: "There is no doubt but that these forces (which were active in his case) were intermittent, like the orthodontic forces used by Oppenheim." In the use of strong forces, as which continuous forces have to be considered, and which were used by Stuteville, Skillen, and others, this formation never can be created, and therefore has never been observed. When the periodontal tissue is crushed by the use of strong forces, no vessel cushion can be formed nor osteoclasts, and the locally necrotic bone is removed after weeks of osteoclastic activity from the neighborhood by undermining resorption. In these places of undermining resorption and sometimes of concurrent undermining resorption of the cementum, a great amount of hard material is removed. The local pressure is accordingly diminished and, under these circumstances, nature does not find it necessary to form this protective cushion of vessels. Before the undermining resorption produced this space, on the very point of the crushed tissue no reaction at all could take place—no formation of osteoclasts, much less of vessels. The same process repeats itself when the tooth movement does proceed, still under the influence of these strong forces.

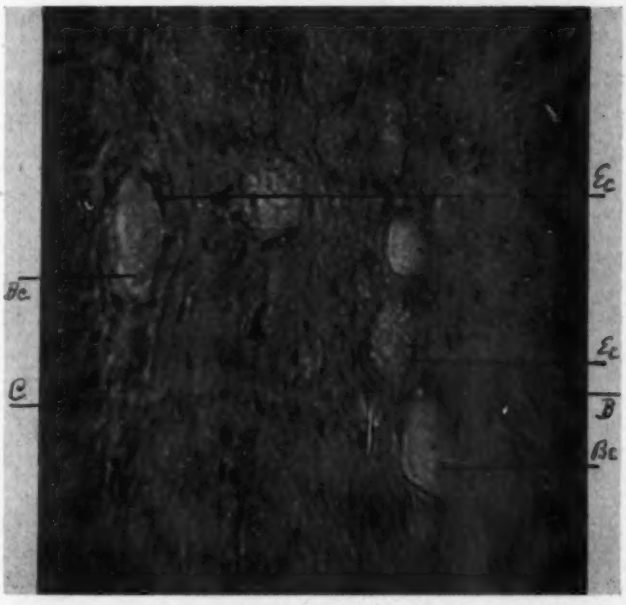


Fig. 6, Specimen No. II.—*V* of Fig. 5 in high magnification; *Ec*, endothelial cells; *Bc*, blood corpuscles; *B*, bone; *C*, cementum.

This cushion formation may also be considered as a peculiarity of the human periodontal membrane and there only under certain conditions already mentioned. The increased number of vessels favors and encourages, without doubt, the resorption of bone and as Kronfeld[29] says: "This assumption is supported by the observation that bone resorption is usually associated with increased vascularity and abundant blood supply."

It was also suggested by a prominent pathologist that we may have to deal in these instances with some kind of fibroid degeneration as described by Hopewell-Smith[20] who considers a "areolisation" of the periodontal membrane as an integral constituent of this degenerative process. I cannot admit that

both these processes are of the same nature. As characteristic for the fibroid degeneration, Hopewell-Smith[20] points out: ". . . in addition they (these spaces) do not possess the definitive walls of arteries, veins, or capillaries," and are often found in the enlarged marrow spaces of the bone. Furthermore, "all traces of the osteoblasts have vanished absolutely, a few decrepit nuclei alone indicating their anatomical positions in the membrane; and there are no epithelial rests of Malassez. . . ."

Just the opposite is found in our specimens: the vacant spaces possess the characteristics of vessels, the endothelial cells (*Ec* in Figs. 6 and 22), and sometimes they contain blood corpuscles. We find osteoblasts on the bone surface (Fig. 5*Ob*); we find the nuclei of the fibroblasts well stained (Fig. 5*p*, and Fig. 22*F*), and we find the epithelial rests of Malassez (*M.c.* in Figs. 5 and 21). All these findings indicate that we have to deal with living and reactive tissue and not with some kind of fibrous degeneration. We see further that this formation, unlike in Hopewell-Smith's cases, is not generally distributed within the whole periodontal membrane but is strictly limited to the areas of increased pressure; it is a local formation.

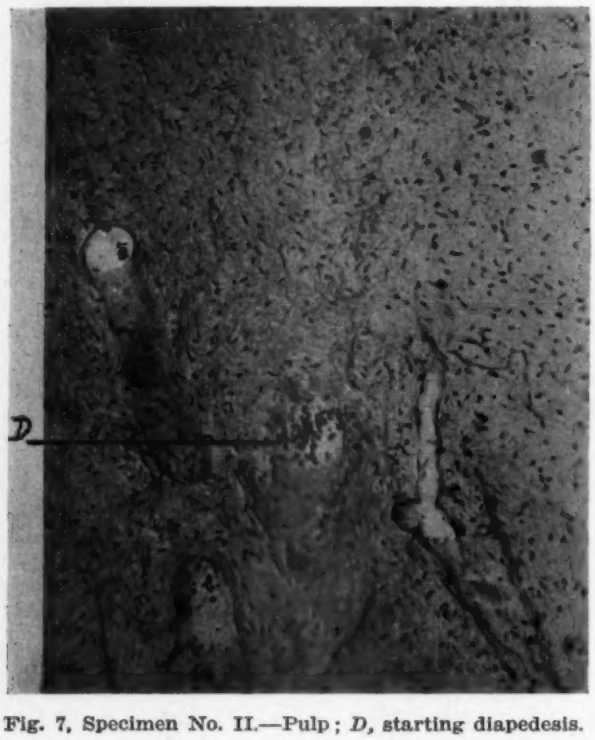


Fig. 7. Specimen No. II.—Pulp; *D*, starting diapedesis.

As we have to deal with the initial stage of vessel formation, we may accept that they follow also here the rule that "they always arise by budding from pre-existing blood vessels"; that they "at first are solid accumulation of endothelial cytoplasma" (Maximow[33]) which "later becomes hollowed out from within" (Schaffer[52]). "First they are devoid of blood cells and are empty" (Maximow[33]).

Let us now return to the description of our specimens. In Fig. 7 we see a part of the pulp of Specimen II in higher magnification, and we observe the

diapedesis of blood corpuscles just starting (Fig. 7*D*), caused by the increased pressure in the veins on account of some compression and tugging at the apex rendering more difficult the back flow of the blood. This is only a confirmation of Krogh's[32] statement that, "where the blood flow is low and the capillaries generally dilated, the process of diapedesis can be observed. . . ."

Specimen III (No. 80). This tooth is a maxillary premolar of the same patient as Specimens I and II; it was moved buccally for four days by a spring of stainless steel 0.2 cm. thick, 1½ cm. long and soldered to a lingual arch. Although some bone was removed with the tooth, it was dislocated and injured during extraction. Nothing can be said, therefore, about the bone and the conditions of the periodontal membrane. Fig. 8 is an outline picture. The apical foramen is still open. The crown was moved to the left, and a deviation of the apex occurred in the opposite direction. As a sequel we find two cementum resorptions near the apex at *R*, Fig. 8. These are reproduced in higher magnification in Fig. 9. Only scanty multinucleated osteoclasts are to be found (Fig. 9*Oc*). At *p* we see the remnants of the periodontal membrane. In the pulp, no peculiar changes are to be noted except many hemorrhages which are to be seen as small dots (Fig. 8*d*); but these may have been caused during the exposure of the pulp before extraction. Special attention will be given to the occurrence of such hemorrhages in future work in order to see whether they are real artifacts or caused by the movement. On account of this uncertainty, these hemorrhages are not reproduced in higher magnification.

Fig. 8, Specimen No. III.—Outline picture; *d*, hemorrhages; *R*, cementum resorptions.

Specimen IV (No. 226) deals with a mandibular first premolar of a 12-year-old patient. A silk ligature applied for twenty-four hours created a separation pushing the crown toward the left side of Fig. 10 which represents an outline picture. The groove near the cemento-enamel junction is an artifact (*A*). Below the artifact we see two distinct cementum resorptions (Fig.

10, *a* and *b*), the lower of which is reproduced in higher magnification in Fig. 11. It has a length of 0.9 mm. No multinucleated osteoclasts are to be seen in this section, and they are also very scanty in the others. The cementum of the remaining root surface in this section is intact. In following the series, we find that the left side of the root surface, i.e., the pressure side, is intact,

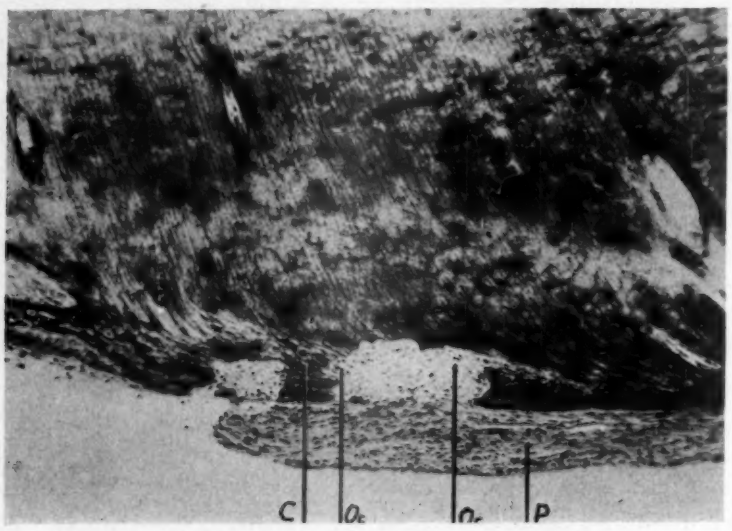
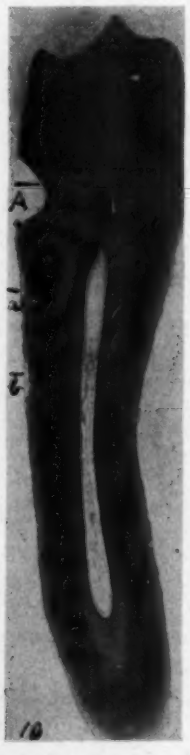


Fig. 9, Specimen No. III.—*R* of Fig. 8 in high magnification; *Oc*, multinucleated osteoclasts; *P*, tissue of the periodontal membrane; *C*, cementum isle between the two resorptions.

Fig. 10.



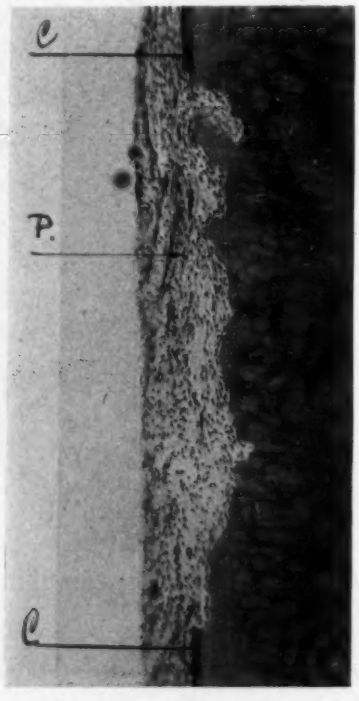


Fig. 11.

Fig. 10, Specimen No. IV.—Silk separation; crown tipped to the left; outline picture; *a*, *b*, cementum resorptions; *A*, artifact.

Fig. 11, Specimen No. IV.—Resorption *b* of Fig. 10; *P*, tissue of the periodontal membrane; *C*, cementum.

while on the previously intact traction side a cementum resorption is found. The silk, being compressible, permitted the tooth to be pushed back by the occlusal forces, causing by this jiggling movement a resorption on the original traction side. This resorption, shown in higher magnification in Fig. 12, has an undermining tendency for, leaving some cementum at its borders intact, it penetrates underneath the cementum (*C*) into the dentine. But it may be suspected that we may have to deal here with a cementum resorption already present before the separation, for the surface is too smooth, and some secondary cementum deposition (Fig. 12*s.C.*) is present. Further experiments of this kind have to prove whether such a jiggling movement with its consequences, i.e., cementum resorptions on both sides of the tooth, may often be found in silk separation, and whether a time of twenty-four hours will be sufficient not only to create the cementum resorptions but also to start the deposition of secondary cementum.

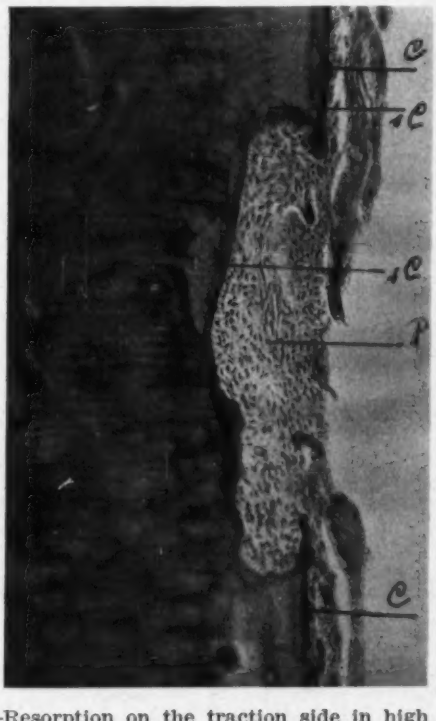


Fig. 12, Specimen No. IV.—Resorption on the traction side in high magnification; *P*, tissue of the periodontal membrane; *C*, cementum; *Sc*, secondary cementum.

In proceeding with the series from the buccal to the lingual, we approach the lingual root surface, and a section here is reproduced in Fig. 13. To the left is the mesial side, and at the lower end of the section the lingual root surface is cut. It is not the apex. We see that the resorptions present on the pressure side in Fig. 10, and disappearing later on, reappear here again (Fig. 13, *a*, *b*, and *c*); the two upper ones are reproduced in higher magnification in Fig. 14, while the resorptions on the lingual side (the "apex") caused by the deviation of force (Fig. 13, *d* and *e*) are shown in higher magnification in Fig. 15. In all these resorptions, practically no multinucleated osteoclasts can be detected.

We see here again how varying the conditions may be in the different parts of the root surface, that a single section does not prove anything, and that only serial sections can give a true picture of the conditions in a given case. Fig. 16 shows the pulp just opposite the resorption *b* in Fig. 10. While the rest of the pulp reveals nearly normal conditions, we find here, confined to a limited space, the sequel of the stasis caused by the interference with the normal blood flow. We find an accumulation of fluid between the pulp tissue elements (Fig. 16*Fl*) and the formation of vacuoles (*V*) among the odontoblasts on both sides. These are dispersed and compressed and have partly disappeared, impairing their ability and the necessity of dentinoid formation. While this dentinoid just above and below the vacuole formation shows the normal width (Fig. 16, *a* and *b*), it is considerably reduced at *c*. Little hemorrhages in the pulp tissue or minor disturbances of the odontoblasts generally are considered

Fig. 13.

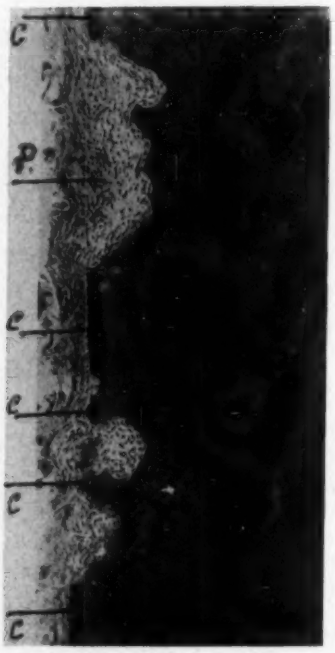


Fig. 14.

Fig. 13, Specimen No. IV.—*a*, *b*, *c*, cementum resorptions on the mesial approximal surface; *d*, *e*, cementum resorptions on the lingual root surface.

Fig. 14, Specimen No. IV.—*a*, *b*, from Fig. 13 in higher magnification; *P*, tissue of the periodontal membrane; *C*, cementum and cementum isles.

as harmless. Orban[44] remarks: "Unimportant bleeding in the pulp or signs of degeneration of the odontoblasts . . . must not be regarded as serious since we know that such conditions can be endured without special consequences." We know, however, that even "minor circulatory disturbances in the pulp vessels cause the formation of calcium salt deposits (calcospherites) and false denticles" (Kronfeld[28]). Because of the lack of collaterals, even minor circulatory disturbances cause circumscribed hyaline degeneration of some connective tissue fibers, which serve as a nucleus for the deposition of calcium salts and the formation of denticles (Willman[65]). While it is well known "that the vacuolar degeneration of the odontoblastic layer is the best predisposition for vacuolar atrophy" (Euler-Mayer[9]), we know, on the other hand,

that the reparative capacity of the pulp is very high and that, even in fractured roots, if no infection intervenes, it not only maintains its vitality (Schier,[53] Brauer,[6] Kronfeld[26]), but is forming new odontoblasts on the spot of the fracture, thus starting to close the gap from the inside by new secondary dentine formation (Euler-Mayer[9]), while on the outside the deposition of secondary cementum contributes to the closure of the defect. Even if a larger amount of odontoblasts is lost, they regenerate from the undifferentiated mesenchyme cells of the pulp (Euler-Mayer[9]).

Fig. 15, Specimen No. IV.—*d, e,* from Fig. 13 in higher magnification.

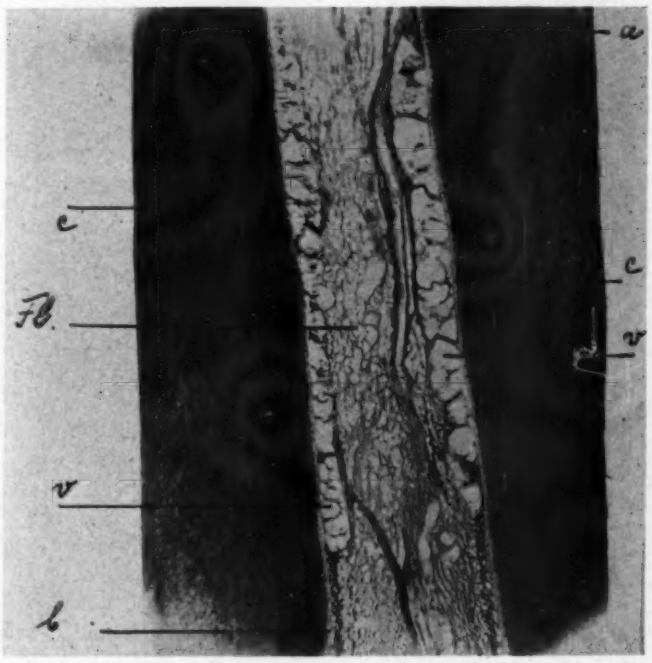


Fig. 16, Specimen No. IV.—Pulp; *Fl,* Fluid accumulation; *V,* vacuoles; *a, b,* normal odontoblasts and dentinoid; *C,* reduced dentinoid formation.

Much has been written about the restorative faculties of the pulp (Euler-Mayer,[9] Orban,[45] Kronfeld,[26] Feldman,[11] Hess,[18] Neuwirth,[36] Rebel,[47] Hopewell-Smith[19]). I will not deny these faculties, at least not for the initial stages, and will admit the different viewpoints of the clinician and the pathologist. What the former would consider as normal and healthy as long as the vitality test proves normal, and no clinical signs of abnormal conditions are present, the latter would consider as pathologic on account of his microscopic findings.

All will depend on the severeness of the injury, the duration of the interference, the patient's age and the individually so different tolerance and resisting power of the pulp. This is especially of importance in the case of hemorrhages. As soon as the escaped blood corpuscles become disintegrated, they render acid the normally alkaline reaction of the pulp tissue, and on the degree and the duration of this acidity depends the whole future of the pulp. A longer duration must cause its death. Fischer[11] expresses it thus: "It is the calcium that decides on the life or death of the pulp." Though the opinions of the various authors regarding the ultimate fate of a previously injured pulp are still quite divergent, it is proved from very extensive research by Euler-Mayer,[9] "that the once initiated atrophy of the pulp may last unchanged for a very long time, but on the whole the condition is on a perpetual decline."

The findings in the pulp by the different authors were based on material, as already mentioned, subjected to the experiment for only a relatively short time. We will see later what can happen after many months of irritation and injury, though I will not and cannot assume that this has to be considered as the general outcome. The material at my disposal is too limited.

Fig. 17.

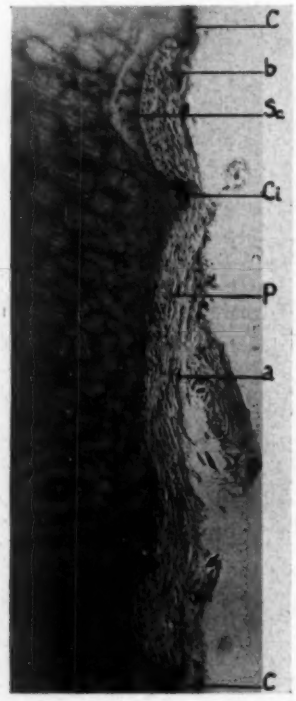


Fig. 18.

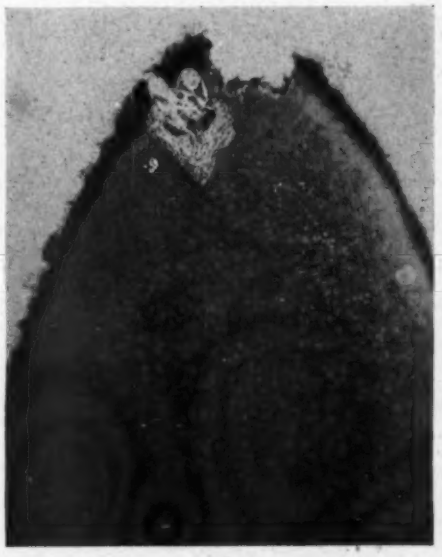
Fig. 17, Specimen No. V.—Silk separation; crown tipped to the right; outline picture; *a*, cementum resorption at the approximal surface.

Fig. 18, Specimen No. V.—*a*, from Fig. 17 in high magnification; *a, b*, cementum resorptions separated by a cementum isle *Ci*; *P*, tissue of the periodontal membrane; *C*, cementum; *Sc*, secondary cementum.

Specimen V (No. 227). This is a maxillary premolar of the same child as Specimen IV; here, also, the force of a separating silk ligature was applied for twenty-four hours. The tooth was sectioned from the buccal toward the lingual. Fig. 17 is a tangential section long before reaching the middle of the tooth; the upper end is therefore not the apex, but some region between the upper and middle third on the buccal side of the root. The crown was pushed to the right,

and we find correspondingly a cementum resorption on the pressure side at Fig. 17*a*. This resorption, shown in Fig. 18 in higher magnification, consists of a lower larger (*a*) and an upper smaller (*b*) resorption separated by a remnant of cementum (Fig. 18 *ci*). While the lower part is in an active state of resorption, the upper one shows already the signs of repair, i.e., the deposition of secondary cementum (Fig. 18 *Sc*) to a considerable degree. The same suspicion arises here as in relation to Fig. 12, i.e., that this resorption (*b*) may have existed even before application of the force. There is another question that, for the time being, has to remain without answer—the time required in man to create resorptions or to repair them. We are not certain of anything about this, though we can adduce some information in this paper relative to the first question. But here again, the great individual differences have to be taken into consideration. The "apical" region in Fig. 17 was devoid of any resorption; but in Fig. 19 we find two resorptions caused by the deviation of the force toward the buccal. In both

Fig. 19.



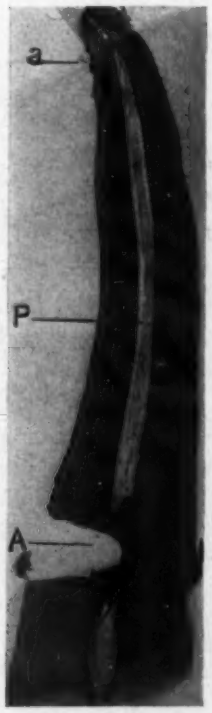


Fig. 20.

Fig. 19, Specimen No. V.—Neighboring section of Fig. 18; by the deviation of force cementum resorptions have been developed on the lingual root surface.

Fig. 20, Specimen No. V.—Outline picture; on the pressure side near the apex cementum resorption *a*; *A*, artifact; *P*, pulp.

resorptions the multinucleated osteoclasts are quite scarce. Proceeding in the series, we find the outline picture Fig. 20, where the deep hole on the cementoenamel junction on the left is an artifact. The pressure side on the right toward which the crown was moved is free from any resorption, but we find a small one in the pressure region on the opposite side near the apex (Fig. 20*a*). This is reproduced in higher magnification in Fig. 21. We find an acute state of resorption without any multinucleated osteoclasts, but the cushion of vessels found here again in its initial state (Fig. 21*v*) may exert some osteoclastic activity. This

cushion is shown in high magnification in Fig. 22. There is no doubt about the presence of endothelial cells (Fig. 22*Ec*) in the borderline of these "vacant spaces." Compare these with the endothelial cells *Ec* in Fig. 40 in the blood vessels of the pulp. The presence of blood corpuscles cannot be distinctly ascertained, but it is beyond any doubt that these formations are not artifacts. Following the series toward the lingual, we find the continuation of resorption *a* in Fig. 17, only much more extended. It remained shallow and did not advance very much in depth (Fig. 23). We see the shallow resorption progressing somewhat deeper only at *c* where it is interrupted by two cementum isles (*a* and *b*). These more resistant cementum isles disappear later also. The accumulation of multinucleated osteoclasts can be seen for the first time in this specimen at *oc* in Fig. 23, and in high magnification in Fig. 24. They are somewhat lifted from the lacunae formed by their activity and are separated from the tissue of the periodontal membrane at the right (Fig. 24*p*) by an artificial gap (Fig. 24*A*). No explanation can be given for the accumulation of these multinucleated osteoclasts just in this locality. In most of the other specimens of short force application, they are found only rarely or not all. The cementum resorption at the apex (Fig. 20*a*) increases in depth and extension as we follow the series.

Fig. 21.

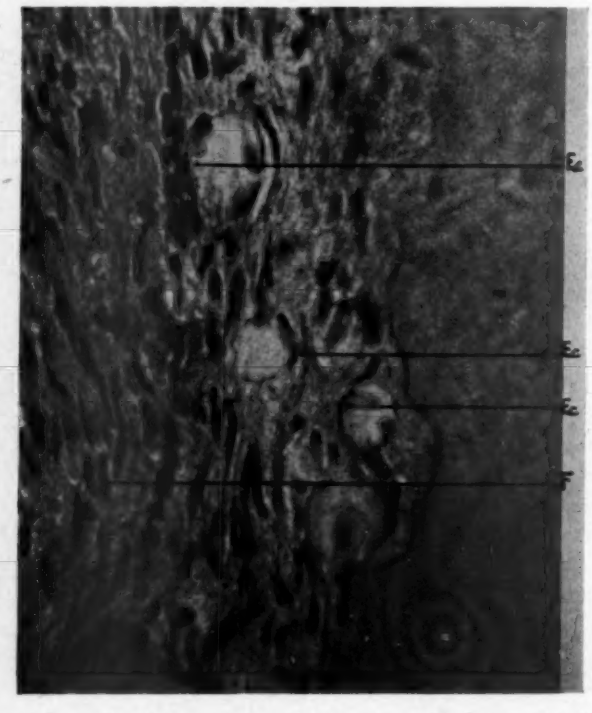


Fig. 22.

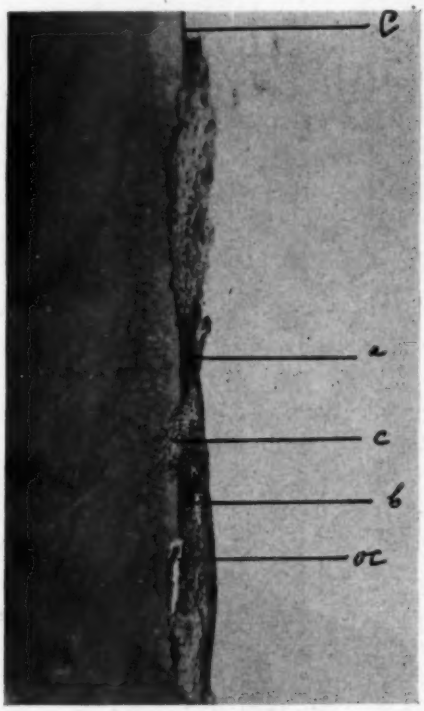
Fig. 21, Specimen No. V.—*a* from Fig. 22 in high magnification; *P*, tissue of the periodontal membrane; *C*, cementum; *V*, cushion of vessels; *M.c.*, epithelial rests of Malassez.

Fig. 22, Specimen No. V.—*V* from Fig. 21 in high magnification; *Ec*, endothelial cells; *F*, well-stained cells of the periodontal membrane.

The cementum resorption in Fig. 23 can be traced without interruption in 128 sections, each one being 0.016 mm. thick with a buccolingual extension of 2.04 mm. (0.016 × 128). Then follows a stretch of 1.5 mm. of normal cementum (96 sections of 0.016 thickness) which, toward the lingual, is followed again by a resorption of 1.14 mm. width (78 by 0.016).

Assuming, according to different textbooks, that the average buccolingual width in the third of the root near the cemento-enamel junction is 7 mm., we see that 3.18 mm. (2.04 + 1.14 mm.), nearly half the width, are consumed by resorptions. The length of the resorption in Fig. 23 is 2.75 mm. The pulp corresponding to *P* in Fig. 20 shows on the right side a normal odontoblastic layer and predentine (Fig. 25*Pr*) of normal width, while on the left we see many vacuoles (*va*) between the odontoblasts, dispersing and compressing them, thereby accordingly reducing their ability to form predentine which is, therefore, much thinner here (*R*). Adjacent to these vacuoles, we find an accumulation of fluid between the meshes that are greatly enlarged by this plasma fluid (Fig. 25*F*). The vessels (*V*) in the longitudinal and cross sections do not contain any blood corpuscles.

Fig. 23.



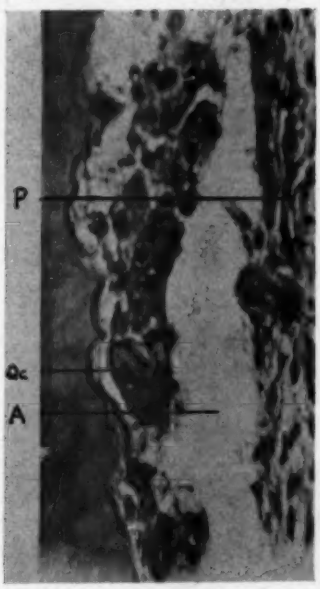


Fig. 24.

Fig. 23, Specimen No. V.—The same resorption as Fig. 18; *C*, cementum; *a*, *b*, cementum isles; at *c* the resorption is somewhat deeper; *Oc*, row of multinucleated osteoclasts.

Fig. 24, Specimen No. V.—Osteoclastic row *Oc* from Fig. 23 in high magnification; *P*, tissue of periodontal membrane; *Oc*, multinucleated osteoclasts; *A*, artifact.

Specimen VI (No. 224). This specimen, a maxillary premolar, is from the same patient as Specimens IV and V. The force was applied for twenty-four hours by wire separation in the routine way. Fig. 26 is an outline picture; the deep hole at the right of the cemento-enamel junction is an artifact. By the wire separation, the crown was pushed to the right which, therefore, is the pressure side. A shallow resorption reaching from *a* to *b* (Fig. 26) is to be observed; it has a length of 1.56 mm. and can be followed from buccal to lingual without interruption in 240 sections, each section being 0.016 mm. thick. The width of the resorption amounts to (0.016 by 240) 4.3 mm., more than half of the width of the root. At the opposite side near the apex in this section, no resorption is to be seen, while it is plainly demonstrable in the neighboring areas. A part of the

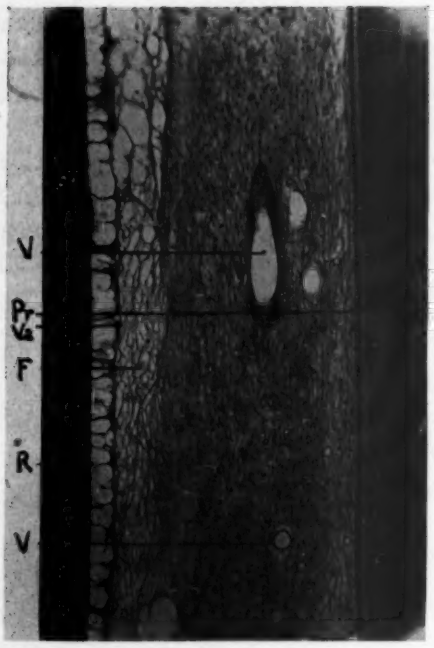


Fig. 25.

Fig. 26.

Fig. 25, Specimen No. V.—Pulp; *F*, fluid accumulation; *V*, vessels; *Va*, vacuoles; *Pr*, predentine; *R*, reduced predentine.

Fig. 26, Specimen No. VI.—Wire separation; crown tipped to the right; outline picture; on the pressure side a shallow resorption from *a-b*; *A*, artifact.

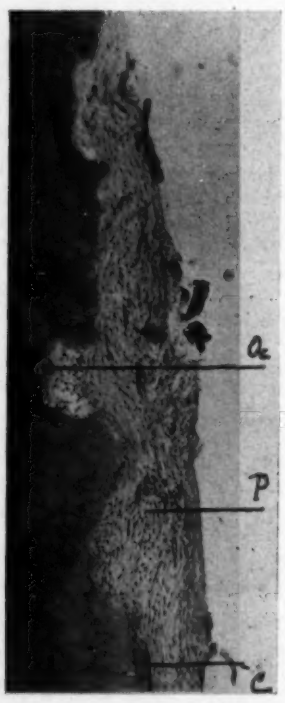


Fig. 27.

Fig. 28.

Fig. 27, Specimen No. VI.—Cementum resorption *a-b* from Fig. 26 in higher magnification; *C*, cementum; *P*, tissue of the periodontal membrane; *Oc*, multinucleated osteoclasts.

Fig. 28, Specimen No. VI.—Another section in the continuation of the series; the cementum on the pressure side on the right does not reveal any damage; on the pressure side near the apex a deep resorption *R* has developed.

resorption *a-b* Fig. 26 is reproduced in higher magnification in Fig. 27, taken from a neighboring section. It extends upward in the picture (toward the root), and although somewhat shallower is twice as long. Very few multinucleated osteoclasts can be seen (Fig. 27*oc*). In following it up, we find near the end of the series the outline picture as reproduced in Fig. 28. The right side, i.e., the pressure side, does not reveal any damage of the cementum, but at the pressure side near the apex we see quite a deep resorption (Fig. 28*r*) that is reproduced in high magnification in Fig. 29. Very few multinucleated osteoclasts as at *Oc* can be seen. The resorption is quite deep and approaches the root canal at the deepest point (*d*) within a distance of 0.09 mm.

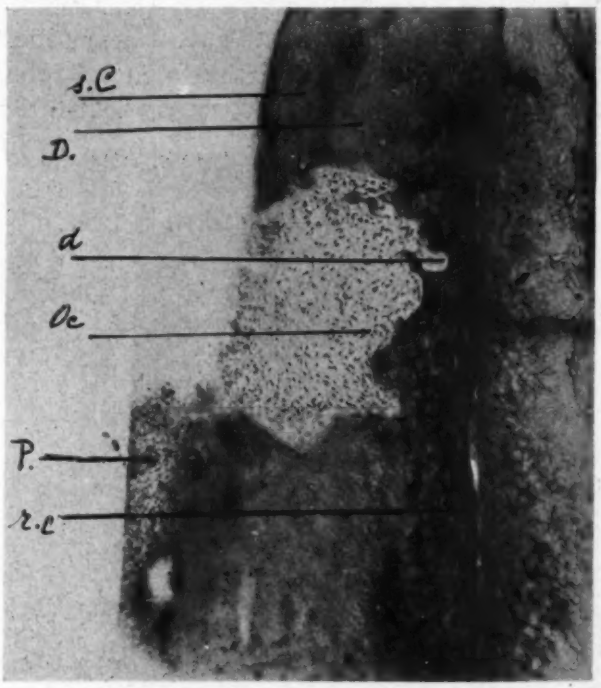


Fig. 29, Specimen No. VI.—Cementum resorption *R* from Fig. 28 in high magnification; *sc*, secondary cementum; *D*, dentine; *rc*, root canal; *Oc*, multinucleated osteoclast; *P*, tissue of the periodontal membrane.

The root wall in this region measures 0.46 mm. The depth of the resorption at point *d* measures 0.37 mm., so that only 9 hundredths of a millimeter of dentine covering the pulp was left. In one of the next sections the resorption is more extended and still deeper (Fig. 30). *R* is the root canal and, at the deepest point, *d*, only four hundredths of a millimeter of dentine covers the pulp. The root canal may be assumed to have a central position as estimated from its location just at the lower border of the picture. If we assume, therefore, the left root wall including the secondary cementum to be of the same thickness as the right one, we have the following numbers: wall thickness at the right side corresponding to "d" 0.18 mm. The remaining dentine at *d* measures 0.036 mm. The depth of the resorption is, therefore, 0.144 mm.

It must be admitted that the root end in this specimen is tapering to quite an extraordinary degree, restricting the wall thickness accordingly.

Specimen VII (No. 225). This is a mandibular premolar from the same patient as Specimens IV, V, and VI. The force applied for twenty-four hours

was that of a separating wire. The outline picture is to be seen in Fig. 31. The excavation on the right at the cemento-enamel junction is an artifact. The crown was pushed to the left and the apex deviated to the right; by this tipping, two pressure zones were created, one on the left below the alveolar crest, the other one on the right near the apex. The resorptions caused by this movement are seen at *a, b,* and *c,* Fig. 31. The resorption *a,* requiring no special attention, is not reproduced in higher magnification. It can be followed without interruption in eighty-eight sections. Sixteen sections of normal cementum follow and then the resorption starts again and can be followed through 178 sections. Each section being 0.016 mm. thick, the corresponding numbers for the width of the resorption in a buccolingual direction are: first, resorption (88 by 0.016) 1.6 mm.; then a strip of intact cementum (16 by 0.016) 0.3 mm. wide, and last the widest part, (178 by 0.016) 3.26 mm. Including the thin strip of intact cementum, the resorption measures buccolingually 5.16 mm., nearly four-fifths the width of the

Fig. 30, Specimen No. VI.—In continuing the series the resorption shown in Fig. 29 is more extended and still deeper; *C,* cementum; *P,* tissue of the periodontal membrane; *R,* root canal.

root (7 mm.). The length changes continuously and measures in some places 1.70 mm. The resorption *b,* Fig. 31, is shown in higher magnification in Fig. 32, *P,* exhibiting an undermining character and coming quite near to the pulp. At the thinnest point at *a,* Fig. 32, the thickness of the dentine amounts to 0.018 mm. The resorption *c,* Fig. 31, some distance above *b,* is reproduced in higher magnification in Fig. 33. It has a length of 1.9 mm. At *a* and *b,* Fig. 33, two cementum isles withstood the resorption, but at *d* it penetrated the dentine and came near to the pulp (*r* c), leaving only a layer of dentine 0.09 mm. thick. No multinucleated osteoclasts can be detected. If we follow the series from buccal to lingual, the picture changes. On the pressure side near the alveolar crest the resorption *a* from Fig. 31 is still to be seen, accompanied by a second one on a somewhat deeper level (Fig. 34 *b, c*). They are not reproduced in

higher magnification. The two resorptions near the apex *b, c,* Fig. 31, have disappeared, and instead we find a new one at *a,* Fig. 34. This resorption is magnified in Fig. 35. The pulp is opened, and the signs of acute resorption can be seen at *a, b, c* (Fig. 35). Some periodontal tissue remained adherent (*d*) and is in close contact with the pulp tissue (*p*). Not only the elements of these two tissues, but also their staining differences stand out quite remarkably. Some fibroblasts of the connective tissue of the periodontal membrane are merging already with the pulp tissue (Fig. 35*f*), and may bring about a regressive metaplasia if the perforation will be of a longer duration, replacing the highly differentiated pulp tissue with connective tissue of a lower function, capable of forming bone or cementum but not dentine. It may be assumed also that the resorptions in Fig. 32 and at *d* in Fig. 33, having approached the pulp within 0.018 and 0.09 mm., respectively, would have perforated into the pulp within a few more hours of continued pressure. The pulp resembles very much the pulp reproduced in Fig. 25 except that no edema was found. Within the odontoblasts, some vacuoles are interspersed, and at these places one finds a reduced width of the predentine. Some vessels seen do not contain any blood corpuscles. Otherwise the pulp tissue seems to be normal.

Fig. 31.

Fig. 32.

Fig. 31, Specimen No. VII.—Wire separation; outline picture; crown tipped to the left; *a*, cementum resorption and two other ones at the apex *b* and *c; A*, artifact.

Fig. 32, Specimen No. VII.—Resorption *C* from Fig. 31 in high magnification; *P*, tissue of the periodontal membrane; *Rc*, root canal; *C*, cementum.

We have seen the vast and sometimes destructive changes brought about by increased pressure within the periodontal membrane. This apparently insignificant membrane, as already pointed out, is of the utmost importance for, and deserves the greatest consideration of, the orthodontist, more than any other tissue; for he has to rely exclusively on this tissue of but one-fifth of a millimeter

thickness for all the changes that have to be brought about in order to obtain the desired results. "There is only one controlling structure, one dictator, and this is the peridental membrane" (Skillen[55]). We must, therefore, try by all means to preserve the vitality and readiness for reaction of this tissue which proves so extremely sensitive to the least interference. It should be emphasized once more that all our successes and failures depend on a tissue of only 0.2 mm. thickness—a most important fact that often is not kept in mind. On this tiny structure depends the fate of the denture almost entirely.

Fig. 33.

Fig. 34.

Fig. 33. Specimen No. VII.—Resorption *C* from Fig. 31 in high magnification; *C*, cementum; *a*, *b*, cementum isles; *P*, tissue of the periodontal membrane; *Rc*, root canal.

Fig. 34. Specimen No. VII.—*b*,*c*, Two small resorptions on the pressure side and one near the apex at *a*, perforating into the pulp.

But there is another factor of the same importance: the preservation of the bone of the alveolar process. The anatomic peculiarity of this structure is generally not given due consideration. As we know from the fundamental work of Loos,[32a] which has already been referred to and which was also illustrated in this journal at another occasion,[38] the buccal and labial plates of bone against which the teeth are moved in a great percentage of our cases consist only of a very thin layer amounting to half a millimeter, which toward the alveolar crest becomes often razor-edged. The obstacle to our movements, the bone, has to give way. This can happen only by osteoclastic activity. It is, therefore, no wonder that this thin bone will often disappear entirely when teeth are moved for distances that exceed, in most cases, many times the thickness of the bone. But nature has provisions for counterbalancing this thinning and disappearing. This compensating measure involves the formation of the so-called osteophytes on the outer surface of the bone, while it is being resorbed from the inside. The formation of

osteophytes is very well developed in dogs and monkeys, but very poorly in man. This was demonstrated by the writer on another occasion.[39] These osteophytes need much time for their development, and we have to furnish them an opportunity by proceeding slowly. Otherwise the bone disappears to a great extent by undermining resorption. This is extremely detrimental if it happens at the alveolar crest. A probably permanent damage may thus be inflicted, as has been pointed out in the beginning. To recollect, we have to deal with two factors: the very thin tissue of periodontal membrane and the quite limited thickness of bony plate. This fact calls for a careful and slow procedure.

If we omit this precaution, failures result which become apparent after years when we no longer see our patient and which the general practitioner generally does not trace back to orthodontic treatment. These cases are superficially diagnosed and classified as pyorrhea. For the failure occurring a shorter or longer time after completion of treatment, i.e., for the relapses, other contributory causes have to be held responsible; but this lies beyond the scope of this treatise.

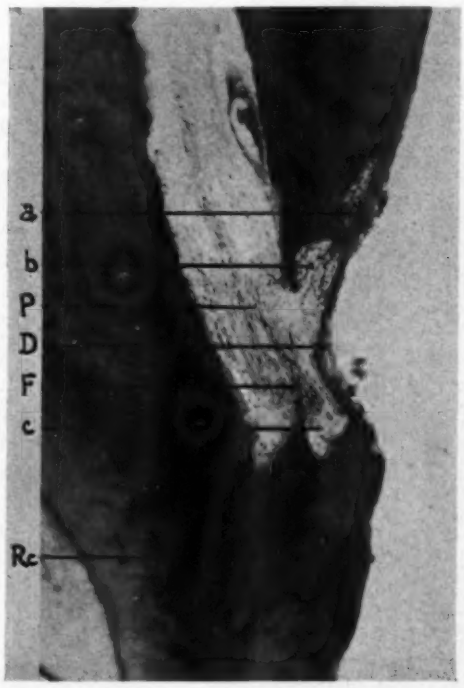


Fig. 35, Specimen No. VII.—Resorption *a* from Fig. 34 in high magnification; *P*, pulp tissue; *D*, tissue of the periodontal membrane; *F*, some cells of the periodontal membrane merging with the pulp cells; *a,b,c*, resorptions in an acute state opening the pulp.

The four specimens (IV, V, VI, and VII) in which the separation was performed in the routine way are from the same patient, and were given to me by a professor of orthodontics of one of our leading universities, from his private practice. These four premolars were extracted in order to gain room for the retraction of the front teeth in a case of double protrusion. During this movement, one of the canines died, an occurrence hardly ever observed by this orthodontist in similar cases. The basal metabolic rate proved to be −24, and this may have been responsible for the lowered tissue tolerance in this case. The metabolic rate is another question that needs closer attention and investigation,

as well as the question of diet, vitamins, and endocrines, though in this field already some important research has been performed. (Becks[2a, b] and others.)

The next two specimens were obtained from an orthodontist, and were removed after different periods of treatment. The teeth were extracted without any bone adhering.

A quite striking difference observed between these two teeth and the four of short intervention which also were simply extracted is that in the specimens of short interference a much greater amount of periodontal tissue was adherent to the extracted teeth. As all these teeth were derived from a young patient of approximately 12 years of age, it may be assumed that, in the four cases of short interferenee, the attachment of the fibers to the cementum was stronger than their tensile resistance; they were, therefore, torn during the extraction, while in the two cases of longer interference the tensile resistance of the fibers was greater than their attachment to the cementum. The fibers were pulled out from the cementum perhaps on account of its somewhat lowered vitality, which according to Gottlieb[13] changes according to the conditions present.

Fig. 36.

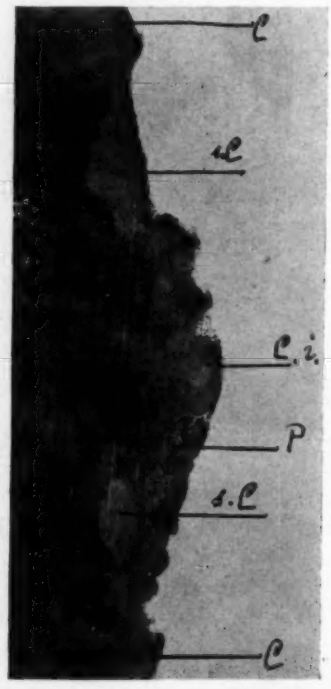


Fig. 37.

Fig. 36, Specimen No. VIII.—Cross section; outline picture; one resorption at the distal surface from *a-b;* another one at *c,* and a quite extended one at the linguomesial corner from *d-e; V,* vessels in the pulp.

Fig. 37, Specimen No. VIII.—Resorption *a-b* from Fig. 36 in higher magnification; *C,* cementum; *Ci,* cementum isle; *sC,* secondary cementum; *P,* tissue of the periodontal membrane.

Specimen VIII (No. 199). This case concerns a maxillary premolar of a 12-year-old child, who revealed a hypothyroid condition. The tooth was not in occlusion; the appliance consisted of a lingual arch and intermaxillary elastics in order to move the buccal segments distally. The treatment lasted three years. The tooth was sectioned crosswise. A section somewhat below the cemento-enamel junction is shown as an outline picture in Fig. 36. In the linguomesial corner

the resorption extends from *e* to *d*. On the distal approximal surface we find two resorptions, a larger one extending from *a* to *b* and a smaller one at *c*. In other sections we find also resorptions on the buccal and mesial approximal surfaces that remained intact in this section. In higher magnification, resorption *a-b* is shown in Fig. 37, revealing an isle of cementum (*cI*) that withstood the resorption. The resorption *e-d* in Fig. 36 is magnified in Fig. 38. The deposition of secondary cementum (*s C*) is far advanced. For higher magnification, the resorption *c*, Fig. 36, is taken from a neighboring slide; here it is still in a subacute state that is reproduced in Fig. 39, while all other resorptions of this tooth are, as we have seen, in an advanced state of repair. No osteoclasts can be found, and in only one place (Fig. 39 *s.C.*) secondary cementum deposition has started. The pulp, that in general shows no severe retrogressive changes, aside from widely dilated blood vessels (*v*, Fig. 36), displays in many places diapedesis of blood corpuscles, the danger of which was outlined above. Such an area of diapedesis is shown in high magnification in Fig. 40. *Bc* indicates the extravasated blood corpuscles.

Fig. 38.

Fig. 39.

Fig. 38, Specimen No. VIII.—Resorption *e-d* from Fig. 36 in higher magnification; *C*, cementum; *sC*, secondary cementum; *P*, tissue of the periodontal membrane.

Fig. 39, Specimen No. VIII.—Resorption *c* from Fig. 36 at another level in an acute state; *C*, cementum; *P*, tissue of the periodontal membrane. *SC*, secondary cementum.

Specimen IX (No. 79). This specimen is a maxillary premolar of a 13-year-old child treated for eighteen months, with an edgewise arch. No further details in relation to the history of the case were obtained. It is a pity that of all the cases shown and also of this one no x-ray pictures were taken, to enable us to combine and compare both the x-ray and the histologic investigation. This specimen was also cross sectioned starting from the apex. One of the first sections near this region is shown as an outline picture in Fig. 41. At the buccal

side, on top of the picture, the whole root wall is resorbed away exposing the remains of the pulp (p). On the resorbed dentine surface, secondary cementum (Fig. 41$s.C.$) is laid down in patches. On the opposite, the lingual side, approximately one-half or one-third of the root wall is resorbed, and secondary cementum

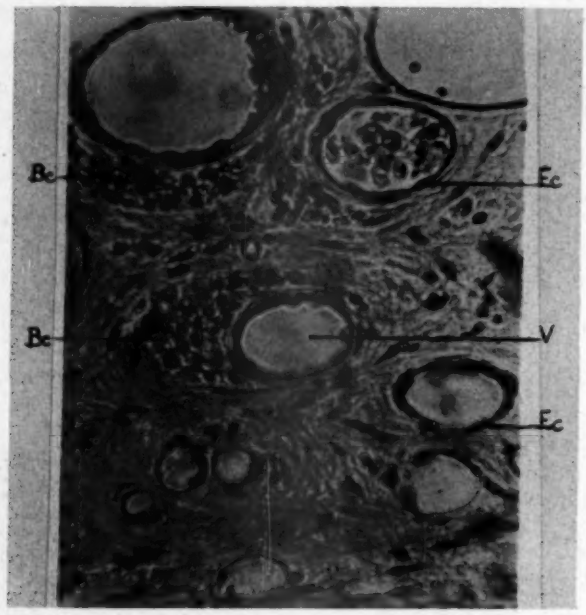


Fig. 40. Specimen No. VIII.—Pulp; *Bc*, diapedesis of blood corpuscles; *V*, vessels; *Ec*, endothelial cells.

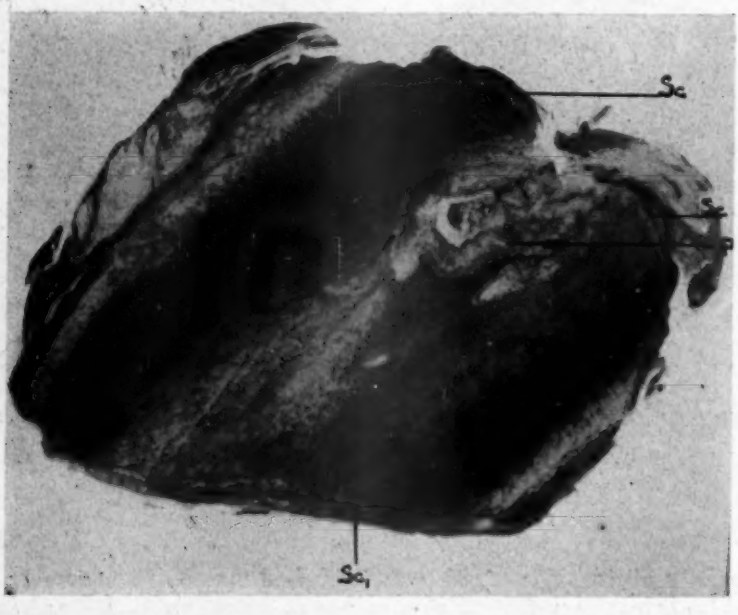


Fig. 41. Specimen No. IX.—Cross section near the apex; outline picture; buccal wall, on top, entirely resorbed away and pulp, *P*, exposed; on the lingual a great part of the root wall resorbed and covered with secondary cementum Sc_1; *Sc*, patches of secondary cementum buccally.

($s.C_1$) in thick layer covers the whole surface. In the exposed root canal, reproduced in high magnification in Fig. 42, only minute degenerated pulp tissue is to be seen (Fig. 42p), consisting of connective tissue which by its products,

cementum, and bone, filled up the entire root canal (*r*). No traces of any odontoblasts can be found in the whole series.

In some places, the resorption was proceeding from the inside of the root canal into the dentine (Fig. 42*a*), but here also the process is at a standstill. These canals are filled with cementum-bonelike structures. At *s.C.*, Fig. 42, secondary cementum is deposited on the dentine surface. At *E*, Fig. 42, we find a termination of an epithelium proliferation that has grown down to near the apex from the mouth epithelium, rendering impossible any reattachment of the periodontal fibers to the root surface, which, of course, can only happen when cementum is laid down first. This lack of reattachment is responsible for the permanent looseness of the tooth; but, at the same time, the stimuli of function cannot be transmitted any longer to the bone and, as an inevitable consequence, the alveolar bone will gradually but surely disappear. In these cases, a functional repair can never occur.

In proceeding toward the crown, we find the condition as shown in Fig. 43. Here where the root diameter is somewhat wider we see that on the buccal side (on the top), approximately half of the root wall is resorbed, while on the lingual side, the resorption has taken away only a part of the

Fig. 42, Specimen No. IX.—Opened root canal from Fig. 41 in high magnification; *r*, calcified pulp tissue; *P*, connective tissue replacing the remnants of the pulp; *sC*, secondary cementum; *a*, resorption canals into the dentine filled out with bone-cementum-like structures; *E*, proliferated mouth epithelium.

root. Here the resorbed surface is entirely covered by secondary cementum (Fig. 43 *s, C.*), while on the buccal only here and there a small amount of this tissue can be found (Fig. 43 $s.C_1$). In the region *a*, Fig. 43, the resorption is still in a subacute state. The unequal resorption on the buccal and lingual sides can roughly be estimated by the seemingly eccentric position of the pulp canal. But it is only eccentric because on the buccal surface much more tooth substance has been resorbed away. At *E*, Fig. 43, we find the epithelium the termination of which we have already seen in *E*, Fig. 42. This epithelium prevents the deposi-

tion of secondary cementum as seen in *D*, Fig. 43, where the exposed dentine did not receive a new cementum cover. More closely toward the gingival margin, the cross section represents itself as seen in Fig. 44. While on the lingual surface, only a few small resorptions in the state of repair are present (Fig. 44*r*), we see on the buccal, at the top, the exposed dentine without any trace of secondary cementum. At this level, the resorptions on the buccal and lingual sides are relatively small, but we find large ones on the mesial and distal approximal surfaces, both in the state of repair (Fig. 44 *a* and *b*). The resorption *a* is reproduced in higher magnification in Fig. 45, while *b* is shown enlarged in Fig. 46. We see the fairly advanced formation of secondary cementum at *s.C.* in Figs. 45 and 46. At C_1 Fig. 46, an isle of the primary cementum resisted resorption. The pulp near the cemento-enamel junction is represented in Fig. 47. The root canal is filled up entirely with a bone-cementum-like structure. Just in the center there remained some pulp transformed by retrogressive metaplasia into connective tissue. No trace of odontoblasts can be detected in the whole series.

Fig. 43.

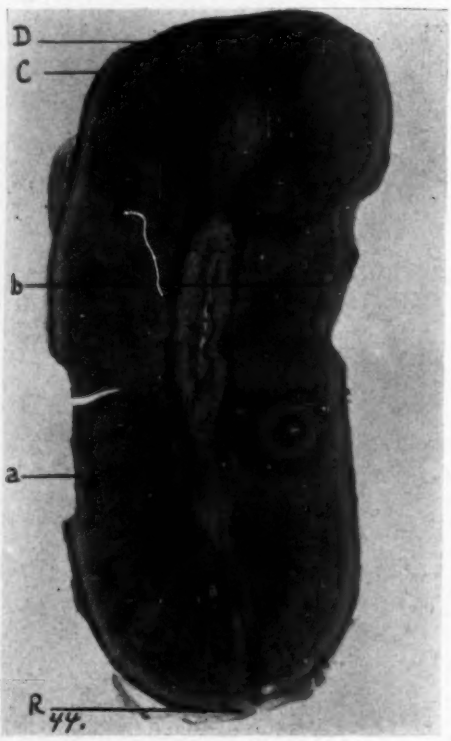


Fig. 44.

Fig. 43, Specimen No. IX.—Cross section between the lower and middle third of the root; *Sc*, secondary cementum; *D*, exposed dentine on the buccal side covered only with patches of secondary cementum Sc_1; *a*, subacute state of resorption; *E*, proliferated mouth epithelium.

Fig. 44, Specimen No. IX.—Cross section in the upper third of the root; *C*, cementum; *D*, exposed dentine not covered with secondary cementum on the buccal side; *R*, three cementum resorptions with two cementum isles between; *a* and *b*, cementum resorptions on the approximal surfaces.

All these morphologic changes in the periodontal membrane as well as in the cementum and in the pulp cannot be looked upon as theoretical damages; they are real and sometimes lasting damages which surely are not apt to prolong the life of the tooth, and which certainly cannot be considered as an appropriate price for the benefit of the treatment. This report gives further evidence of the many damages which are inflicted by artificial forces applied for short or long

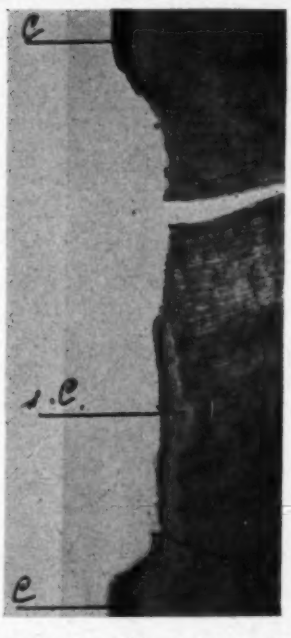


Fig. 45.

Fig. 46.

Fig. 45, Specimen No. IX.—*a* From Fig. 44 in higher magnification; *C*, cementum; *sC*, secondary cementum.

Fig. 46, Specimen No. IX.—*b* From Fig. 44 in higher magnification; *C*, cementum; C_1, cementum isle between the two resorptions; *sC*, secondary cementum; *P*, tissue of periodontal membrane.

Fig. 47, Specimen No. IX.—Pulp; with the exception of some connective tissue in the center replacing the pulp tissue *P*, the root canal is filled up with a bone-cementum-like structure *BC*; no trace of any odontoblasts.

periods of time. The author does not stand alone in this assertion. We find in the literature several reports which corroborate these findings and statements. Skillen,[55] for instance, speaks of "rather severe injuries which arise during treatment" and at another occasion[56] he maintains "that changes in the periodontal membrane may occur that are not recoverable."

I cannot agree with Grieve[14a] when he says: "Greater progress might possibly result if those men with biologic tendency would place a higher value upon knowledge gained through clinical experience, for from this source we have learned considerable." I think that the time when our knowledge and advance was purely empirical is over. A more or less mechanically oriented attitude should be considered obsolete, and the mechanics and procedures should be adjusted to conform with the results of scientific findings as is the routine way in other scientific branches. Theory paves the way for practice. If men with a "biologic tendency" have also some experience in the practical field of orthodontics, so much the better. They can combine theory and practice and prove, or try to prove, their interdependence.

This the author has tried to do for thirty years, and he has been benefited thereby. He does not say that, by doing so, he could avoid failures altogether, but he could sometimes surmount difficulties and avoid a few otherwise certain failures. If we will apply to our practice the additional theoretical knowledge gained by this human material, then one of my previous statements can be repeated once more with still greater justification, namely that we have to deal with very delicate tissues which cannot be treated too gently.

CONCLUSIONS

1. Light forces are to be used, and frequent rest periods allowed.

2. By all means at our disposal, the right diagnosis has to be made before treatment is begun. The movements once started can then be carried on continuously in the same direction. There will be no need then to change the diagnosis and consequently change the direction of movement. The damages inflicted will thus be far less and surely will help to improve the final result.

It is not the purpose of this paper to frighten anyone. Orthodontics will be done and has to be done; but we have to be aware of the bare facts that govern our work and cannot be avoided. Knowing these facts and their causes, we will exert the utmost care in the future.

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TREATMENT OF TEMPOROMANDIBULAR DYSFUNCTION ACCOMPANIED BY SEVERE PAIN SYNDROME

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THIS case is illustrative of a severely painful dysfunction of the left temporomandibular joint, wherein the principal etiologic factor was an anomalous right condyle whose defect was reflected in abnormal jaw development and in an anomaly of occlusion.

On Aug. 12, 1940, the patient, a native born woman of Turkish descent, aged 22 years, was referred to the orthodontic clinic from oral surgery, with a complaint of severe pain in the left temporomandibular region.

Medical history revealed that the patient had had whooping cough, mumps, and measles. The approximate dates of onset and the duration of these illnesses were not available. The patient's tonsils were removed when she was 5 years of age. There was no history of birth injury, trauma, or of recent illness.

Clinical intraoral examination revealed a normal mucosa and gingiva. The tongue was tapering and of normal proportions. The teeth were of apparently normal size and enamel texture. Several fillings were noted. Oral hygiene was good. In the upper arch all the teeth were present, with the exception of the third molars. In the lowar arch three incisors were missing, having recently been removed due to their involvement in a radicular cyst.

Movement of the mandible was limited in both vertical and lateral excursions. The maximum opening in the right canine area was 18 mm., and 12 mm. between the maxillary left second molar and mandibular left first molar. The mandible appeared to be underdeveloped. The right side of the face showed normal contour, while the left side presented a flattened appearance. In going through its allowable vertical motion, the mandible swung to the left. The left condyle was very prominent, while very little motion could be detected in the right condyle during jaw movement. All mandibular movements were painful to the patient.

DESCRIPTION OF PAIN SYNDROME

Approximately ten months prior to presenting at the orthodontic clinic at Columbia University, the patient developed a severe pain in the left temporomandibular joint. From this point the pain radiated to the ear and temporal region. Although the pain was at first intermittent, it later became constant and more severe when jaw movements were attempted. Forced movement, as in yawning, brought on excruciating paroxysms. The patient claimed that the pain was more intense during cold, damp weather. She could not lie comfortably on her left side for any length of time.

Gnathostatic diagnosis revealed the following: (Fig. 1) In its relation to the median plane, the upper denture showed a total, extreme, dental and alveolar

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Read before the New York Society of Orthodontists, Nov. 11, 1941.

contraction. In the lower, the incisal area showed a mild dental and alveolar contraction, while the lateral segments showed a mild dental and alveolar distraction. The discrepancy between the right and left halves of the denture should be noted in the upper right hand corner of the chart.

In its relations to the orbital plane, the denture (Fig. 2) showed a dental and alveolar protraction in the lateral area of the upper. The degree of protraction in the right lateral segment was greater than in other parts of the upper. It was also noted that the distance between the prosthion and the orbital plane was the accepted "norm" of 5 mm. The lower denture showed a total dental, alveolar and mandibular retraction. This, together with the extent of the facial deformity, is readily noted in the gnathostatic photographs (Fig. 3).

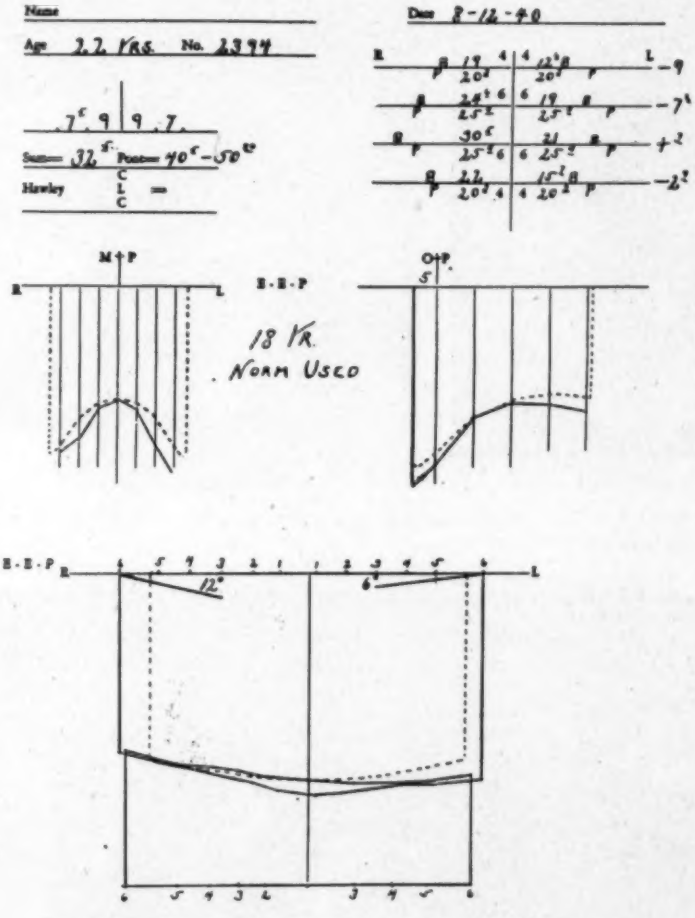

Fig. 1.—Chart of first gnathostatic diagnosis.

Observations on the relations of the denture to the horizontal Frankfort plane gave some indication as to the nature of the temporomandibular dysfunction (Fig. 4). In the upper an abstraction was noted in the incisal area and in the left lateral segment. The lower showed a mandibular abstraction increasing in degree from right to left. Note lower graph in Fig. 1. Gnathostatic models also revealed a displacement to the right of the entire denture.

Roentgenographic projections of the temporomandibular areas proved most interesting and served to shed much light upon the problems presented in this case (Fig. 5). In the closed position, the left condyle was noted to be in a slightly forward relation to normal. This abnormally close relationship of the condyle head to the posterior surface of the articular eminence is due to the deformity present on the right side (Fig. 6). The right side does not present

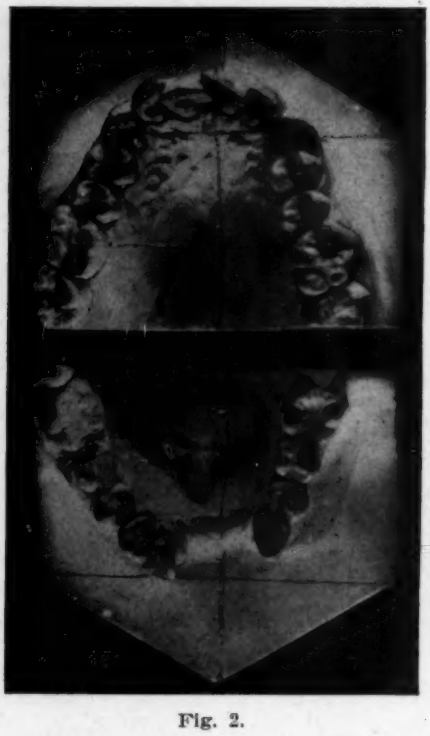

Fig. 2.

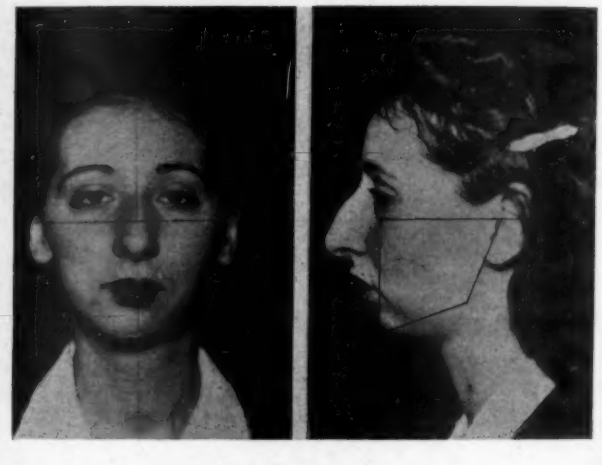

Fig. 3.

Fig. 2.—Occlusal view of models, showing projection of the sagittal and orbital planes.
Fig. 3.—Gnathostatic photographs.

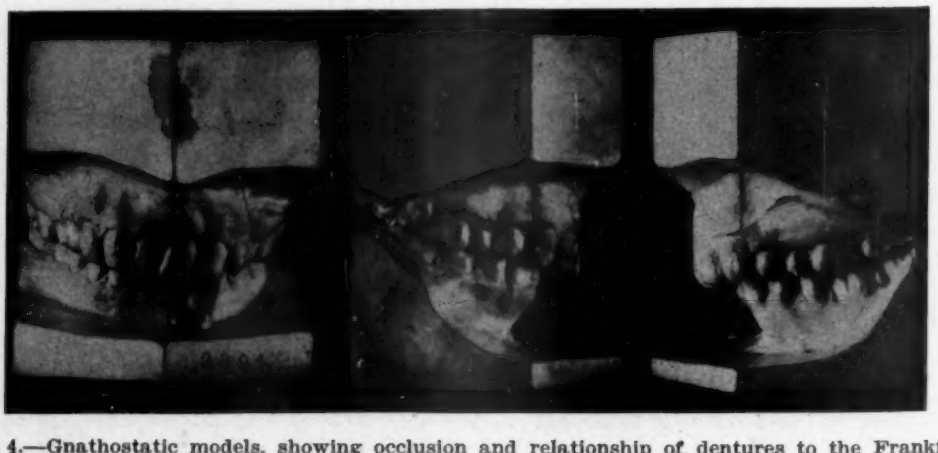

Fig. 4.—Gnathostatic models, showing occlusion and relationship of dentures to the Frankfort horizontal or eye-ear plane.

a condyle head in relation to the mandibular fossa. Instead, there is present only a fragmentary stump of the neck of the condyle. The condyle head appears as a completely detached and encapsulated sequestra, being displaced medially and lying in the region of the sigmoid notch.

That this condition had been operating for some time, was indicated by an acutely bent angle at the juncture of the body of the mandible with the ramus. The molding of the bone was due to an unbalanced force of muscle pull of long duration.

The abnormal position of the left side is further illustrated in the open projection (Fig. 7). Here, with the mouth open to its normal limit of accommodation, the condyle head slides down, just short of dislocation, at the guidance of the slope of the posterior surface of the articular eminence. It is difficult to conceive of a normal meniscus arrangement being present to buffet and accommodate this extreme excursion of the left condyle.

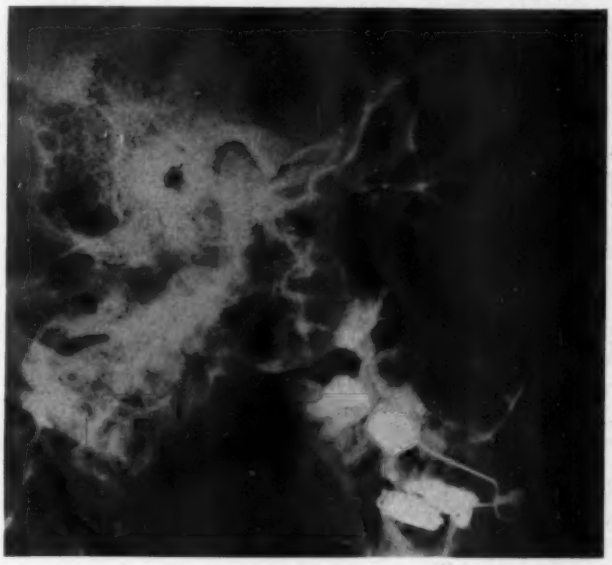

Fig. 5.—Left side, jaws closed.

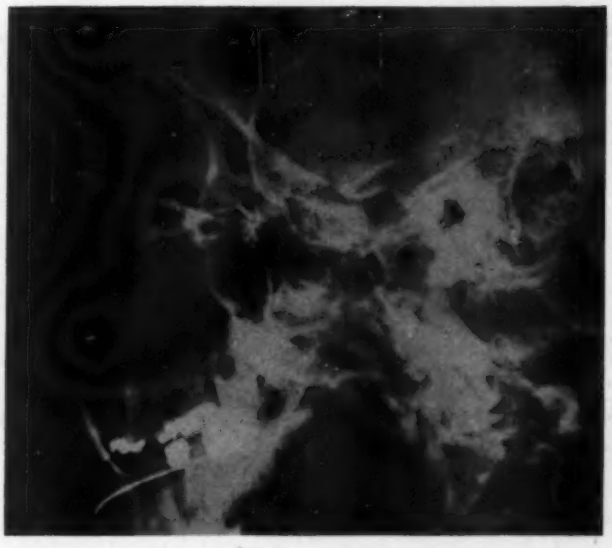

Fig. 6.—Right side, jaws closed.

The open view of the right side (Fig. 8) further illustrates the absence of any condyloid structure in relation to the fossa.

The vertico-submental projection (Fig. 9) graphically illustrates the distortion in contour between the right and left sides of the body of the mandible and further attests to the asymmetry noted in the gnathostatic diagnosis, relative to the relation of the denture to the horizontal plane. It further shows that through a loss of the condyle head of the right side, the lack of support in the mandibular fossa, which permits this side to ride superiorly without the restraining influence of a superior boundary.

Fig. 7.—Left side, jaws open.

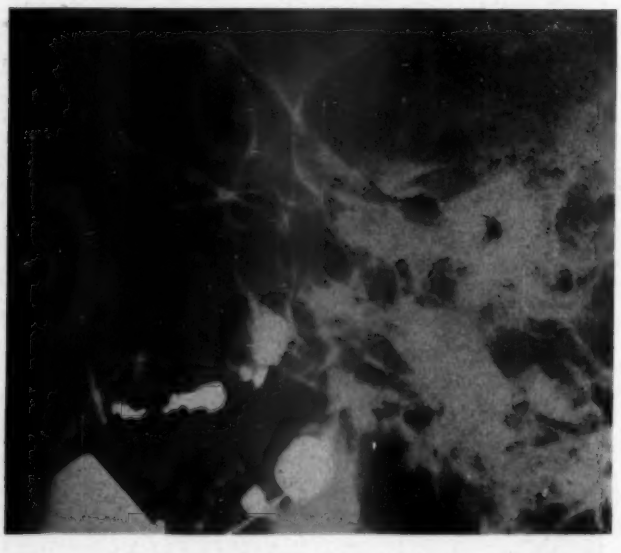

Fig. 8.—Right side, jaws open.

In considering the aggregate picture presented by the roentgenograms, it appears that despite the negative history of birth injury or trauma, the patient, at some time during infancy or early childhood, sustained some form of injury. The injury, in whatever form it may have been applied, was sufficient to cause

a complete detachment of the right condyle from its ramus, and to displace superiorly the normal relative position of the mandibular fossa. The loss of function of the right condyle placed the entire burden of masticatory stress and jaw movement upon the left temporomandibular joint. It is, therefore, believed that the excessive function required of the left temporomandibular joint over a period of several years caused a gradual derangement of the structures which go to make up the joint. Despite the visible facial deformity the patient was free from pain or discomfort until damage to the joint was sufficient to cause the onset of the symptoms previously described.

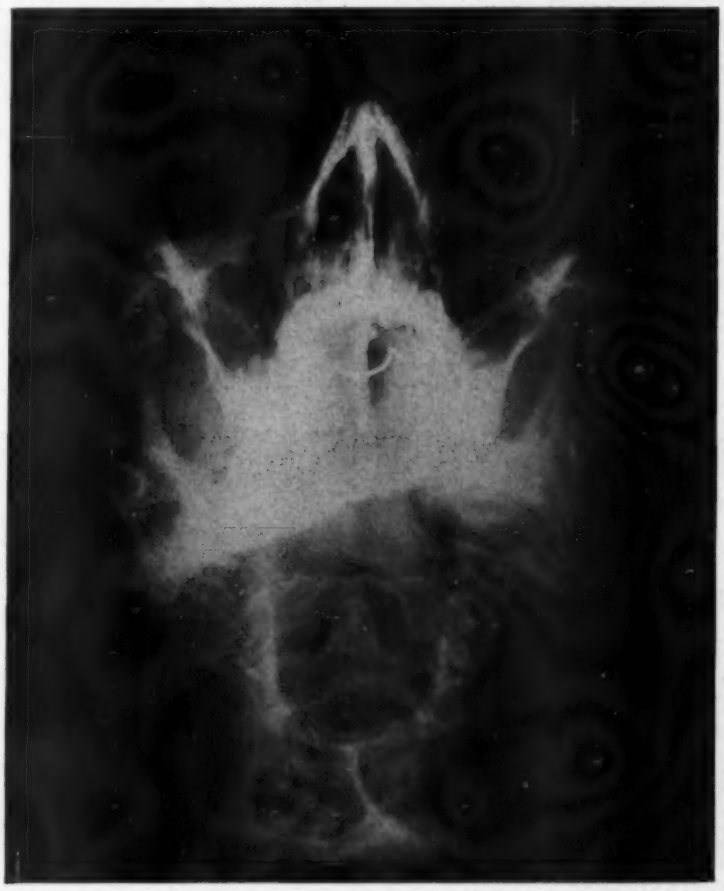

Fig. 9.—Vertico-submental projection, illustrating asymmetry of the mandible.

TREATMENT

Our first step in the treatment of this case was to recommend the removal of a deep mesio-angular mandibular left third molar impaction. This was recommended in order to eliminate any other possible factors which might be contributing to the patient's symptoms. The tooth in question was removed on Sept. 28, 1940. One month later, the patient's first visit since the removal of the impaction, she claimed that the pain was less severe and not quite as constant as previously. She could yawn without the severe paroxysm of pain that usually accompanied such action, but she could not chew comfortably. Mandibular movement was still as restricted as when the patient first presented.

The following month the patient was again complaining of the same severity of symptoms she had had prior to the removal of the impacted third molar. At this time, we decided to construct an appliance which would change the position of the left condyle and alter the direction of muscle forces. This was accomplished in the form of a lower bite block (Fig. 10) built up on the right side.

The bite block was placed in the patient's mouth on Dec. 7, 1940, and the patient was instructed to wear it as much as she could. Wearing the bite block continuously was difficult at first, but as time passed the patient found that she could wear the appliance for longer periods of time with little discomfort.

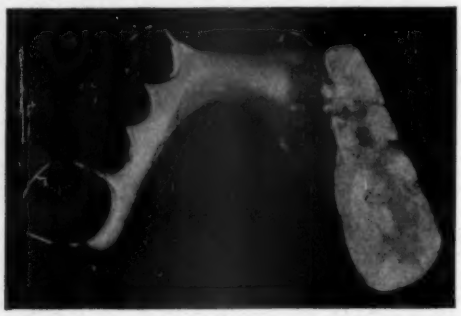

Fig. 10.—Lower bite block.

Fig. 11.—Models of second gnathostatic diagnosis.

By March of 1941, three months after the insertion of the bite block, the patient was able to accomplish wider movements of the mandible. Pain in the left condylar area still persisted, but was less severe. From this point on, the patient showed a rapid and continuous improvement. By April 19, she was quite comfortable, and the range of excursive movements continued to increase. On April 26, she was free from pain.

On May 24, the patient was sufficiently comfortable to become interested in esthetics by expressing a desire to have replacements for the missing mandibular anterior teeth. It is interesting to note that at this time her maximum opening in the right canine area was 28.5 mm. and 19.5 mm. in the left molar area. This in contrast to the 18 mm. and 12 mm. opening when she first presented.

In June of 1941, after the patient had been wearing the bite block for seven months, gnathostatic impressions and photographs were taken again. The findings were as follows: In its relation to the median plane the upper showed a total dental and alveolar contraction, extreme in degree in the incisal area and medium in the lateral area. In the first gnathostatic diagnosis, the upper was in extreme contraction in both the incisal and lateral areas. The lower remained unchanged, presenting a mild dental and alveolar contraction in the incisal area and a mild dental and alveolar distraction in the lateral area.

In its relation to the orbital plane the upper presented a dental and alveolar protraction in the lateral area. The lower presented a total dental, alveolar and mandibular retraction. These findings are the same as those noted in the first gnathostatic diagnosis. The photographs, however, indicated a change in the position and relation of the mandible since the first gnathostatic photos were taken.

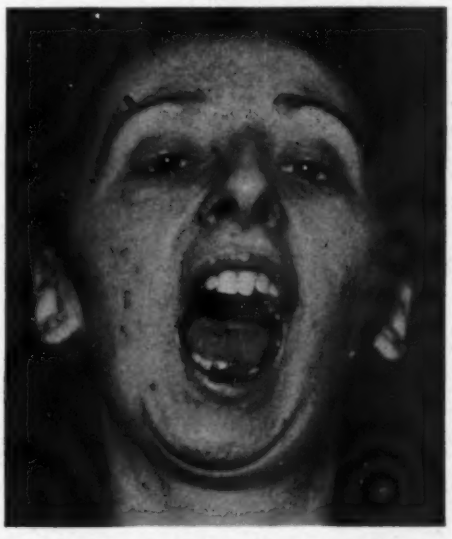

Fig. 12.—Illustrating patient's ability to open her mouth comfortably to the extent of 30 mm. in the right canine area and 24 mm. in the left molar area.

Considerable change was noted in the relation of the denture to the horizontal plane (Fig. 11). The upper presented an incisal abstraction, while the lateral areas showed an attraction on the right side and an abstraction on the left. The attraction in the right lateral area was not so marked in the first charting. The lower showed an attraction in the right and an abstraction in the left lateral areas. These findings are also indicative of a change in mandibular position.

With the accomplishment of our first purpose, that of relieving the patient of the symptoms previously enumerated we decided to institute some form of appliance therapy which, through orthodontic tooth movement, would aid in increasing the vertical dimension. On July 14, 1941, a plain lower lingual arch was placed in the patient's mouth and adjusted for the uprighting of the mandibular left first molar which was tipped mesially. The patient returned three weeks later. Since wearing the lower lingual arch she had not been able to wear the bite block and once again she felt some discomfort in the left tem-

poromandibular area. Impressions were then taken for an upper bite block which was placed in the patient's mouth two weeks later on August 25. At this time she is wearing the bite block nights and intermittently during the day. She is comfortable, happy, and free from pain. Her maximum opening in the right canine area is 30 mm. and 24 mm. in the left molar area (Fig. 12). Jaw movements are accomplished without discomfort.

The absence of a functional right condyle will probably require at some time in the future the use of some form of prosthesis to retain the left condyle in its new position of comfort. This would probably be in the form of a cast appliance to replace the missing mandibular anterior teeth, and carrying at the same time bite-raising overlays for the posterior teeth.

I wish to express my sincere thanks and appreciation to Doctor Sidney Riesner for so kindly assisting with the radiographic aspect of this case and to Doctor Arthur Totten for his kind assistance in treatment planning.

5922 Bergenline Avenue

PRESENTING A NOT SO UNUSUAL BUT FREQUENTLY UNRECOGNIZED TYPE OF CASE IN WHICH ORTHODONTIC THERAPY WAS INSTITUTED FOR THE RELIEF OF PAIN

ELMER H. BROWN, D.D.S.,* AND EUGENE J. KELLY, D.D.S., TRENTON, N. J.

DR. BROWN:

IN MY service at St. Francis Hospital, in Trenton, we have, as all other hospitals and clinics have, many patients that suffer from various symptoms and are regarded as neurotics. Treatment has consisted of optical refraction, septum and antrum operations, and physiotherapy. But they still complain of sinus pains, whatever they are; pains in the triangles of the neck, and pains in the frontal or parietal areas. They ultimately find their way to the dental clinic hoping to discover dental foci of infection. By placing the fingers in the ears while the patient is opening and closing his jaws tenderness of various degrees is usually present, cracking in the joint, and frequently, the inability of the patient to clear the eustachian tube by swallowing.

Study models are necessary in all these cases and orthodontic consultations are very desirable. The case history presented today is not uncommon but unusual in that tenderness was so marked in the temporomandibular articulation that the husband who is a physician was able to eliminate the possibility of nose and throat factors and present a more reliable history than that which is usually available.

Dr. Kelly has not completed orthodontic treatment but as far as I am concerned the case is finished; all symptoms have disappeared, and the patient is happy.

The patient, Mrs. H-----, aged 29 and mother of three children, was a healthy, robust individual who presented a malocclusion with a noticeable lack of development in the vertical relation of the lower third of the face. There were noticeable scars on the neck following cervical adenitis supposed to have been bovine tuberculosis at age of 8 years.

She gave a history of abscessed ears from a very early age, not otitis media, but chronic bilateral, furunculosis of the auditory canals (external otitis). Previous to her first pregnancy her ears had been opened twenty-five times. In 1932 I removed the impacted third molars without relief of symptoms. It was believed at that time that the ear condition was caused by the pathology about these teeth.

From the onset of the menstrual cycle either one or both ears would abscess at time of menstruation. After the first child was born she had no more abscesses but complained of slight earache for approximately two to four years. This condition increased steadily in intensity until it became necessary to administer sedatives nightly. Her husband, being a physician, had eliminated the usual causative factors. Her nose and throat consultations were the best

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Read before the New York Society of Orthodontists, Nov. 10, 1941.

obtainable. It was in desperation that the husband suggested a dental consultation, hoping to find a focus of infection. Routine examination, including radiographic pictures, proved negative on this point; the various excursions usually performed by the mandible were impossible due to a locked bite. It was suggested to the husband, at this time, that extraction of all the teeth or orthodontic treatment was necessary to relieve that trauma present in the fossa. It appeared that this trauma had been a predisposing cause to the abscesses which occurred about the hair follicles. Also, it appeared certain that this same trauma was the cause of the so-called earaches existing at this time.

Fig. 1. Fig. 2. Fig. 3.

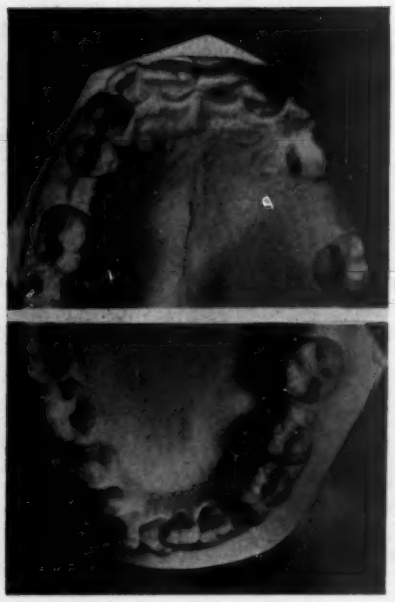

Fig. 4.

Fig. 5.

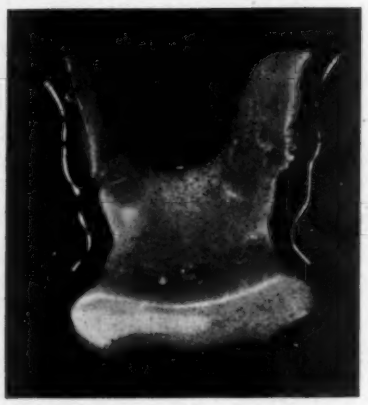

Fig. 6.

It is my opinion that this type of case is common, that is temporomandibular trauma. Rarely, however, is the pain localized; it is usually distributed about the temporal or occipital areas. It should be part of a dental diagnosis to inquire as to any pains not accounted for and to supplement dental x-rays by study models. Any irregularity in occlusion should be given proper consideration.

It is, therefore, apparent that the field of oral diagnosis must include study models and the services of an orthodontist should be available to hospitals for consultations.

Dr. Kelly will follow with a technical description of the orthodontic treatment to date.

DR. KELLY:

A preliminary survey of this case was made on March 15, 1941. It was classified as a mutilated Class II, Division 2 malocclusion (Figs. 1 to 5).

Tie-bracket bands were immediately made and cemented on the maxillary right and left first premolars. Molar bands with tubes for an edgewise mechanism on the buccal surface were also made and cemented at the same time. Size .030 wire was curved to fit the lingual surface of these bands so as to provide an attachment for a bite plate.

At this time an impression of the maxillary jaw was taken. From the subsequent model a bite plate of the type advocated by Dr. Robert Strang for Class II, Division 2 malocclusion was made and placed in the mouth a few days later. This plate, made of acrylic material, opened the bite approximately one-quarter inch in the incisal region and was ground to fit the occlusion of the mandibular anterior teeth (Fig. 6). The patient was dismissed for two weeks with instructions to wear the bite plate at all times except when eating.

Fig. 7.

Fig. 8.

Fig. 9.

Fig. 10.

Inasmuch as most adults whom I treat are not as cooperative as children, I felt that some concession should be made for the sake of comfort, hence the instructions to remove the bite plate at mealtime. This procedure worked out quite well, and when I next saw the patient, she had no complaints other than that for two days or so, when she first worse the bite plate, it interfered with her speech.

All the pain in the region of the ear stopped after she had worn the bite plate two days.

Further treatment consisted of banding the remaining maxillary teeth and aligning them to proper arch form and eliminating the unevenness of the occlusal surfaces (Figs. 7 to 10).

In the latter part of July the patient and her family spent a month at the shore, and I was unable to see her. Approximately a week before she returned the original bite plate no longer fit comfortably due to the shifting of the maxillary teeth. She discontinued wearing it and the day before she returned home, pain again manifested itself in both ears. Just as soon as a new bite plate was made and inserted the pain again disappeared.

Treatment at the present time consists of expansion and alignment of the maxillary, and alignment of the mandibular arches, plus continued wearing of the bite plate to stimulate vertical development in the buccal segments.

This treatment will unlock the bite and enable the patient to perform properly all mandibular excursions as well as give her continued relief from pain.

When orthodontic treatment is completed restorations will be made to insure proper vertical height and relieve the condylar strain.

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Department of Orthodontic Abstracts and Reviews

Edited by

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All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. J. A. Salzmann, 654 Madison Avenue, New York City.

Observations on the Malformation of the Head in Mongoloid Deficiency: By Clemens E. Benda, M.D., Wrentham, Mass., *J. Pediat.* **19**: 800, 1941.

Mongolism is easily recognizable in the age group from 2 to 15 years. There is, however, a definite lack of diagnostic signs which would decide the question in doubtful borderline cases. The main stigmas are epicanthal folds, slanting palpebral fissures, fissured lips and tongue, short hands with curved little fingers, short feet with a gap between the big toe and second toe, stout figure and hypotony, strabismus, deep voice, and a rather typical psychologic behavior, but it is generally agreed that in many cases a part of these symptoms only is present. It always appeared to the author extremely unsatisfactory to use epicanthus and slanting eyes for the establishing of the diagnosis, since both stigmas are not pathognomonic and are less frequent than one would expect them to be.

The so-called "mongoloid" appearance depends upon a growth disorder of the skull which is invariably encountered in every mongoloid child. The mongoloid skull, including the facial bones, deviates from the normal in its proportions, and it is this malproportion of the skull that is likely to decide the diagnosis in doubtful cases. Of course, not only the skull is affected in mongolism; this condition is a general growth disorder which involves the whole skeleton, and this fact is a cornerstone in the understanding of the pathology.

An understanding of the growth disorder of the mongoloid skull will be best reached by comparing it with a normal skull in its development from birth to adult life. To a great extent, anatomists have dwelled upon the changes of the normal skull which occur during the period of development and which are persistent in spite of the encountered variations of familial and racial appearance. In studying skulls, one should always place the specimen in such a position that the so called "base line" or Frankfurter horizontal (a line which runs through the inferior margin of the orbit hole and the superior margin of the external auditory meatus) is in a horizontal position. A perpendicular line is drawn through the alveolar process in front of the face. The skull of the newborn infant differs from that of the adult in several points. Most outstanding in the newborn skull is the large brain cavity and the small face. Average length, measured from the glabella to the occipital point, is 11.5 to 12 cm.; average parietal width (bip.), 9.5 cm. The circumference of the skull is 34 cm., or 13.5 inches. In studying the proportions of the face it is noteworthy that the vertical distance from the vertex to the nasion is twice as large as that from

the latter to the alveolar point. The orbital holes appear disproportionately large in the newborn infant and in comparing the height of the orbital hole with the distance from the lower orbital margin to the alveolar crest, one will recognize that the latter distance measures less than the orbital height. The distance from the anterior nasal spine (acanthion) to the alveolar point (prosthion) is short but large enough to provide adequate berth for the developing upper teeth. A last item of interest is the position of the transverse axis of the atlanto-occipital joint. In the newborn infant it is in the middle of the base line, the proportion between the anterior and posterior sections being 3:3. Growth of the skull base displaces the site of the atlanto-occipital joint backward. The proportion in a 10-year-old child is about 4:3, and in adult life, 5:3. The development of the musculature of the neck counteracts the nodding of the head forward in upright position which takes place if man falls asleep. In the child the head is balanced. During the extensive growth which is going on in the first year of life, the circumference of the skull increases 3 to 4 inches, or 10 cm., the circumference of the head at one year being 42 to 44 cm. In the fifth year of life, the circumference usually reaches 50 cm., or 20 inches. These measurements are of importance because during infancy the skull bones are thin and the sinus system not yet developed. The circumference offers, therefore, a fair estimate of the brain cavity and the size of the growing brain. The increase of the size of the skull is produced by growth of the skull base in the lines of the cartilaginous and membranous synchondroses and by osseous proliferation about the margins of the flat skull bones, the sutures acting like the epiphyseal lines of the long bones. Even more impressive than the increase of the brain cavity is the growth of the facial bones in postnatal life. The distance from the nasal spine to the alveolar crest increases rapidly; the lower orbit margin moves farther and farther apart from the alveolar crest, while the orbit holes gain very little in size. In the adult skull the distance from the nasion to the alveolar point is equal to the height of the forehead. The mandible increases in size, and the mandibular angulation becomes more marked.

Comparison of the mongoloid skull with the normal development demonstrates impressively the failure of an adequate growth of the mongoloid skull. To talk about "the" mongoloid skull is justified by considering the striking resemblance in all of them. The transverse axis of the atlanto-occipital joint in the mongoloid skull is in the middle of the base line in spite of the notorious brachycephaly of the mongoloid skull. This is due to the extreme shortness of the skull basis in mongolism. A second factor is the failure of growth of the maxilla and the nasal portion. The proportion between forehead and face is still fetal. The deficiency of development of the maxilla places the lower margin of the orbit near the alveolar crest and the distance from the nasal spine to the alveolar point is as short as in a normal newborn baby. The incisor teeth have no space for their roots and protrude. The angulation of the mandible is flat and definitely fetal in shape, but its outline is somewhat confused by the fact that the mental process is protruding and the anterior branch is bent; on account of the underdevelopment of the maxilla and the teeth, the mandible does not find the normal counteraction. The strong muscle pull bends the anterior rami of the mandible upward.

In summarizing the abnormality of the facial development of the mongoloid skull, it appears striking that the mongoloid skull is deficient in growth of all those structures which show the most marked development after birth, the nasal bone, ethmoid, and maxilla, which fail to grow, resulting in a persistence of fetal proportions of the face. The outline of the mandible is sometimes confused in later life through the protrusion of the teeth, and prognathism occurs due to muscle traction of the masticatory apparatus, but these secondary changes do not influence the essential feature of mongolism, which consists in the failure of development of the protruding structures, the summits of the face ("acromieria," akros = apex, tip, summit). The micrognathic deficiency is only a part of the picture. The skull basis remains short on account of the insufficient growth of the synchondroses spheno-occipitalis and spheno-ethmoidalis. Another factor which contributes to the shortness of the skull base is the absence of development of the frontal and sphenoid sinuses. By eliminating the sinus system from the skull, one obtains the true outline of a sagittal section through the mongoloid skull. Another factor is the failure of the flat skull bones to produce sufficient growth at the bone margins. This failure is indicated clinically by the slow closure of the fontanels, which remain patent for several years. It is also indicated by the remainder of sutures which disappear normally shortly after birth (frontal suture and sagittal suture) and the creviced sutures at the skull basis. A last item, worth mentioning, is the thinness of the flat skull bones due to failure of diploe development.

It is obvious that mongolism represents a specific growth disorder in which all those structures are involved which grow markedly shortly before and after birth. In a condition which represents the opposite of mongolism, namely acromegaly, the same structures react especially to the abnormal growth stimulation. It is well known that in the latter condition the whole skeleton does not grow indiscriminately. In the skull, for instance, most outstanding is the increased pneumatization which leads to extension of the sinus system. The mandible grows by osseous deposits upon the mental process. In the flat skull bones, the diploe is chiefly involved. On the skull basis, the cartilaginous sutures react especially by renewed proliferative activity of the cartilaginous disks. Mongolism is associated with a deficiency of cartilage proliferation, diploe development, extension of the sinus system, and osseous proliferation about the skull sutures and the protruding parts of the face.

Clinically, the stout, short, and broad fingers and the curvature of the little finger form an important diagnostic item. It is the curvature of the little finger which attracts most interest. The anomaly of the second phalanx is apparently a part of the prenatal growth disorder because the incidence of incurvature of the little finger does not increase with age.

The structures which are expected to increase mostly in size are impaired to the greatest extent, and since the metacarpal bones and the first phalanges have to grow more extensively than the third phalanx, the shortness of those bones is more striking than that of the latter. There is, however, an absolute shortening of all three phalanges, and the thinness of the distal phalanx is an important item which does not appear in the measurement of the length. The

acromicry of mongolism does not depend upon an inferiority of the brain but represents a specific deficiency of the growth factor.

The postnatal mongoloid disorder represents a deficiency of those factors which are generally related to the function of the anterior pituitary. The mongoloid skull at birth has not yet a definitely brachycephalic shape, but the fronto-occipital diameter (normally 11.5 to 12 cm.) appears somewhat shortened, though not in all cases. The width of the skull (biparietal diameter normally 9.5 cm.) is normal or slightly subnormal; the circumference of the skull is, therefore, within normal range but below average (34 cm., or 13½ in.).

Measurements of several mongoloids during the first half year after birth indicated that the skull of the mongoloid infant does not grow in the first few months. This is due to the absence of growth of the skull base. The brain, however, increases in size, and as a compensatory measure, the parietal and temporal parts protrude, producing a "rounding out" of the mongoloid skull which is sometimes so marked that length and width of the skull are equal. At birth the most outstanding peculiarity of the mongoloid child is the face. The bridge of the nose is flat and sunken. Although the nasion is rather small in every newborn baby, the mongoloid shows such an obvious underdevelopment of the root of the nose that this is the most conspicuous sign. Another item is the malproportion of the facial development. The distance between acanthion (anterior nasal spine) and prosthion (alveolar point) is so short that there is only a small crest between the lower border of the nasal cavity and the alveolar edge. The vertical diameter of the orbit holes is definitely larger than the distance from the lower orbit margin to the alveolar crest.

The sphenoid body is smaller than normal and its position is more upright. The distance from the anterior clinoid process to the acanthion is shorter than normal, and the plane of the cribriform plate is displaced to a higher level. Most remarkable is the smallness of the maxilla. The nasal spine is close to the alveolar ridge and the palate appears on the same level as the sella instead of being on the same plane with the skull base. The axis of the atlanto-occipital joint is nearer to the face line than to the occiput, indicating the smallness of the bones forming the skull basis and the facial scaffolding.

In mongolism the fontanels are unusually large and unite after great delay. It has, however, not yet been noticed that all sutures may be separated and that in palpating the skull bones one may find that the sagittal suture is not in approximation and the parietal bones are separated ½ cm. or more. The frontal suture which disappears normally within a few days after birth may be palpated down to the nasion even several months after birth. The same delay is recognizable about the lateral fontanel and those sutures which cross the sides of the skull. This delay or absence of union of sutures may last throughout childhood. Normal growth of the flat skull bones proceeds in proliferative ossification at the margins from one or more centers of ossification. The delay of closure of the fontanels and the open sutures are due to the insufficiency of growth at the margins of the flat bones. These new observations on the skull sutures add evidence to the theory of a deficiency of the growth factor.

These are a few more signs which are of value for the diagnosis and which, in the author's opinion, are caused by the same factors. The skin may be ex-

tremely wrinkled at the wrists and ankles and occasionally at the neck. The skin envelope appears too large for the skeleton which did not keep up to the growth rate of the skin. The same is true for the tongue which may markedly protrude in mongoloid children in later years. The tongue is not too big but the palate is too small for the tongue. It is certain now that the alterations of the tongue in mongolism are secondary changes due to the smallness of the mouth and to abnormal suction.

Benda summarizes that the mongoloid at birth is an infant who suffered from a deficiency of skeletal growth before birth. The prenatal deficiency involves the same structures which are affected in the postnatal period. The deficiency is a "hypomorphy" (Davenport) with a well-defined pathology in which the growth of the distal parts is foremost involved, giving rise to what the author may comprise in the term of "acromicry." The difference between a mongoloid and a normal premature baby is that the latter is proportionate in its features corresponding to the stage of development which it has reached at the time of birth. The mongoloid is not proportionate; it shows a "rounding" of the features because of delay of distal growth. The skull, the hands, the feet, the whole body repeat the same type of abnormality which is due to the fact that the centers of growth are well laid out but the growth slows down toward the periphery and gets arrested.

According to various statistics in different countries, it is estimated that about 2.3 instances of mongolism occur among 1,000 births. Mongolism participated in the production of mental deficiency in about 10 per cent.

An analysis of the factors which produce the peculiar appearance of these patients reveals that the characteristic feature of this condition is due to an abnormal development of the skull. The sphenoid body does not form the normal angulation with the clivus; it is either in an upright position or even slightly bent backward. The plane of the anterior skull cavity is steeper than normal. The cribriform plate is shorter and is placed at a higher level than normally. The nasion is retracted. The growth of the facial bones is diminished. The maxilla body and the mandible are underdeveloped. The development of the sphenoid sinus and of the frontal sinus is either entirely wanting or the appearance of the sinus is delayed and its size is small. The flat skull bones remain paper-thin due to absence of diploe extension. The skull sutures remain permanently separated by fibrous tissue or their ossification is delayed due to the absence of bone proliferation at the margin of the flat skull bones. The closure of the fontanels is delayed for the same reason. The facial development, especially that of the prominent parts of the face, is markedly diminished (acromicry).

An analysis of the growth of the skeleton reveals that the same disorder which is characteristic of the skull affects the whole skeleton. The hand bones are shorter than normal due to a reduced growth rate of the metacarpal bones and the phalanges. Those structures of the hand which increase most extensively in size during life are most severely involved. X-rays of the hand bones indicate smallness and underdevelopment of the distal parts.

Measurements of the heads of mongoloid babies indicate that the skull of patients with mongolism is slightly undersized at birth. X-ray studies reveal a

characteristic deformity of the skull basis and an underdevelopment of the facial structures. An item which has not yet attracted attention is the delay of development of the flat skull bones. The adjacent margins of the bones of the brain case are separated by wide fibrous septa which fail to disappear in time. Metopic sutures are almost invariably found in mongoloids. The observations suggest a deficiency of the growth factor. Anomalies of the hand bones and of the long bones indicate involvement of the whole skeleton. It is evident that the congenital acromicry is independent of the brain of the mongoloid child. Its source has to be sought in agents which are essential during the gestation period and which are apparently connected with the hormonal blood supply.

In order to provide a name for the mongoloid deficiency which encompasses an understanding of the essential feature of this disorder and which permits a broader application to all races, including the Chinese, Japanese, Indian, and negro patients, the term "congenital acromicria" is proposed.

The Genetic and Endocrine Basis for Differences in Form and Behavior as Elucidated by Studies of Contrasted Pure-Line Dog Breeds and Their Hybrids: By Charles R. Stockard and collaborators. With special contributions on behavior by O. D. Anderson and W. T. James. Department of Anatomy, Cornell University Medical College, New York, N. Y. Illustrated with 128 figures, 113 plates and a frontispiece. Pp. 775. Price $7.50, Philadelphia, The Wistar Institute of Anatomy, 1941.

This work is the result of Stockard's use of dogs to test experimentally his views on the endocrinic basis of constitution. A. LeRoy Johnson made observations on the dentition of the different breeds and worked out the indices of the skull.

Familiarity with the exaggerated modifications of growth equips the observer with the basic knowledge for detecting mild degrees of the same expressions which are commonly seen in normal individuals.

A survey of the genetic behavior of the achondroplastic distortion of the extremities is presented, following a more detailed discussion of the findings in cross-breeding of various strains of dogs. Achondroplastic extremities occur as an isolated or localized structural deformity in a number of mammalian species, including man, in otherwise normal individuals. It is suggested hypothetically that a gene of identical chemical nature may exist in widely different members of the animal kingdom and that this gene plays a consistent role in the development of individuals. Head distortions were also found as a strictly localized modification in an animal with otherwise normal structural arrangements.

The skull modifications in certain breeds are accompanied by characteristic arrangements of teeth. Deductions are presented from linear measurements made in different skull types. Little, if any, correlation was found between the linear dimensions of the cranium and the wide variations found in the skeleton of the face.

Cross-breeding is shown to be responsible for discrepancies in pattern between the upper and lower jaws in the mammalian head and that these are

inherited and developed as separate and independent characters and that the foregoing factors underlie the widespread disharmonies of which faulty dental occlusion is most prominent, commonly seen among race and breed hybrids. Abnormal reduction in length of one or both jaws was found to be often associated with chondrodystrophy in the basicranium and other regions. This suggests to the authors that cartilaginous growth plays a more important part in early development and form of the maxilla and mandible than embryologists have generally recognized. The authors conclude, "Finally, the fact that the upper jaw may develop along one pattern, and the associated lower jaw on quite another plan, leads one to question the validity of the theory that their embryonic origin is from different portions of the same branchial arch."

The general shape and size of the jaws and teeth were found to differ among dog breeds, as among human races, and hybrids included many having large teeth in association with small jaws and vice versa. Dogs of hybrid breeding constantly demonstrated structural disharmony and functional maladjustments.

Skull indices obtained in this study indicate that the structural formation in localized regions of the skull determines the type of dental occlusion, "and that it is the facial part of the skull which is of most importance in this connection." The palatal index, as a direct expression of the form of the upper jaw, was found to be more intimately correlated with the nature of dental occlusion than is the index for any other skull feature. Modifications of the mandible in the dog skull were found to be much less pronounced than in the maxilla. "There is considerable probability," we are told, "that similar relations will be found to hold for man, since the disturbances of facial growth in the human infant and child are so readily comparable to the conditions in the skull of the puppy."

This volume will be found of especial interest by those interested in genetics, endocrinology and the problems of growth. Orthodontists who attempt to go beyond the empirical use of mechanical appliances must necessarily belong to the afore-mentioned category. To these practitioners this volume will prove interesting and instructive.

J. A. S.

Editorials

"Speed Up" Orthodontics

Under the head of so-called "rapid treatment," that is to say treatment consisting of two days' to two weeks' duration, orthodontics again makes the headlines in a recent issue of *Time* magazine.

Orthodontic "speed up" treatment as revealed in *Time* is not now interesting or mystifying to the experienced orthodontist; none the less it obviously made the "news."

The experienced orthodontist recognizes so-called "stunt treatment" when he sees it; however, the public does not. He knows the range of application to such treatment is limited entirely to a small fraction of a certain age group of cases, but the public does not. He knows that insofar as the entire field of orthodontics is concerned the treatment described is of little importance one way or the other; however, the public does not know this.

Unfortunately by intimation and implication the reader is led to believe that Yale University, an institution of undisputed high educational standing, has by its clinical research saved 300,000 children annually from painful and costly ordeals; when as a matter of fact nothing new has been contributed not entirely known to the late Edward H. Angle, Calvin S. Case, Henry Baker, and other pioneers of orthodontics as far back as the turn of the century.

Why Yale University and one of its professors should be tied into a story connected with the American Association of Orthodontists meeting at New Orleans will have to be left to the reader's own imagination.

The incident is important at this time because such publicity tends to offset the fine hard work done by the persistent workers of the Bureau of Public Relations of the American Association of Orthodontists during the last few years.

In addition to that it is important because it is plain from the evidence revealed in the mails that many orthodontists throughout the country are being asked why they are unable to perform the "speed up" orthodontic service so recently described in *Time*.

The proper answer to that question would, no doubt, be that orthodontists do not know *how* to do it and neither does the clinic of Yale University, in only exceptional and limited cases; however, that answer would sound much like trying to save face for limitation of ability.

The incident is unfortunate; however, here is probably a place where the Bureau of Public Relations of the American Association of Orthodontists can be of much service in the future in helping lay magazines steer clear of wild "oomph" stories pertaining to the subject of orthodontics.

H. C. P.

Dentists in the Draft

Of no small interest to the dental profession is the policy at this time on the subject of procurement and assignment of dentists to the Army Dental Corps. The action of local draft boards to call dentists for induction into service through the draft has created an important problem in the procurement of dental officers. A situation has been created so that any local draft board by drafting a dentist almost forces the Army to commission the inducted dentist as a dental officer. This situation is plainly causing confusion and paradoxical situations in the procurement of qualified dental personnel for appointment in the Dental Corps.

It is interesting to note that the War Department issued orders in June of 1941 concerning applications of dentists for commission in the Dental Corps who had previously been drafted into the Army. This action followed the hearings held before the Committee on Military Affairs of the Senate in March of 1941. As a result of the hearings the action by the War Department provides a means whereby drafted dentists can secure commissions. It was brought out in the hearings that while physicians and surgeons were able to secure commissions and active duty on a professional status, dentists were unable to do so because it was claimed the dental reserve was sufficient to satisfy the needs of the Army. This meant that all dentists inducted into the military service as privates would not have an opportunity to secure commissions and active duty as dental officers.

It is now presumed that, with the recent completion of the setup known as the Procurement and Assignment Service, complete cooperation between this service and the Army will discontinue the widespread induction of dentists through the medium of the Selective Service, and in this new way dentists will be commissioned, in the future, in accord with the policy of the Surgeon General's office. In that way a complete check on the ability and reputation of the dentist in civilian life will be available for the War Department. It becomes increasingly obvious that the high standard of professional and physical fitness demanded by the Army Dental Corps cannot be reached if the local draft boards are depended upon to "select" dentists who ultimately are to make up much of the Dental Corps of the Army; however, Procurement and Assignment Service changes all of that. Plainly then in the near future all applications for commission in the Dental Reserve, National Guard, and Dental Corps, Army of the United States, will be cleared through the Procurement and Assignment Service before being submitted to the Adjutant General for action.

It is now understood that recommendations have been made to the effect that the Selective Service immediately change their regulations as they may apply to dentists so that no dentists will be called for induction who have not been cleared by the Procurement and Assignment Service and the Surgeon General's office.

The above, no doubt, reflects that progress is now on the way that will tend to remedy some of the paradoxical situations that have been so obvious to the dental profession in the formation of the important Dental Corps of the Army of the United States.

H. C. P.

News and Notes

A. A. O. Officers and Committees

The following men were elected at the meeting in New Orleans:

President-Elect, Archie B. Brusse.
Vice-President, R. C. Willett.
Secretary-Treasurer, Max E. Ernst.

Dr. Claude R. Wood was elected to the American Board of Orthodontics.
The committees for the coming year are as follows:

Judicial Council
Glenn F. Young
R. C. Willett
Joseph D. Eby

Budget Committee
A. C. Broussard
W. A. Murray
Claude R. Wood

Editorial and Publication Board
A. E. Scott
Stephen C. Hopkins
E. C. Lunsford

Public Relations
George A. Barker
H. B. Robison
Henry U. Barber, Jr.

Education Committee
Thomas D. Speidel
George M. Anderson
Harold J. Noyes

Laws and Infractions
J. A. Gorman
Paul G. Spencer
L. M. Waugh
Stanley S. Crouch
John R. McCoy

Research
Milo Hellman
A. G. Brodie
B. Holly Broadbent

Constitution and Administrative By-laws
H. A. Allshouse
Oren A. Oliver
H. C. Pollock

Relief
Harry L. Keel
George H. Siersma
E. B. Arnold

Nomenclature
J. D. McCoy
Russell E. Irish
George R. Moore

Inter-Relations
A. W. McClelland
John W. Ross
George Anderson

Necrology
E. W. Swinehart
A. B. Thompson
Wm. T. Chapman

Program
James W. Ford
Ralph G. Bengston
A. G. Brodie

Local Arrangements
W. A. Murray
G. H. Williams
Howard E. Strange
Leonard Grimson
Howard J. Buchner

Commercial Exhibits
Frederic E. Haberle

Dental Corps Commissions

According to word received from the American Dental Association, the following telegram has recently been sent out by Lieutenant Colonel Sam F. Seeley, Executive Officer of the Procurement and Assignment Service.

"The Office of the Surgeon General has announced that vacancies in the Dental Corps are for the present reserved for only those dentists who are in Class 1A or who have been inducted. I am authorized today to announce that one thousand officers may expect early

commission and that those in Class 1A should apply immediately to the Office of the Surgeon General for application forms stating age, that they are in Class 1A, name, address and school of graduation. All cases will be cleared through procurement and assignment service. Please publicize immediately.''

It is to be noted that this communication differs from the one appearing on p. 260, News and Notes section of the April issue of the JOURNAL, wherein it was stated that to make application for a commission an application should be directed to the Procurement and Assignment Service. In this latest order the application should be directed to the Office of the Surgeon General giving the particulars as related in the telegram. The application will, as is stated, be cleared through the Procurement and Assignment Service but the original contact has been changed to the Office of the Surgeon General.

It should also be noted that only those men in Class 1A should apply. Commissions are reserved for this group of men together with those men who may have been inducted into the service as privates. This, at the present time, does not include those men who registered under the Selective Service Acts on February 16, 1942.

The Scientific and Health Exhibits of the American Dental Association

The Scientific and Health Exhibits which have gradually developed from a minor adjunct to the American Dental Association meetings to a major attraction will take on a new significance this year. Added to the usual colorful exhibits of the educational institutions and individuals, there will be the olive drab and navy blue atmosphere of our armed forces. Things that were formerly of only passing interest to most members of organized dentistry have become highly essential to the very existence of our mode of life. These important new apparatus, instruments, and methods will be readily understood when observed and sketched in the Scientific Exhibits in Boston's historic Mechanics Hall.

Not only are the dental activities of the Army and the Navy now of great importance, but even research and teaching activities are in a process of transition. The large numbers of young men rejected for army duty because of dental defects reveal a need for wide improvements in our methods of dental care and their administration. The teaching, research, and health exhibits will reflect what American dentistry is contributing to American defense.

The increase in interest evidenced by the large and attentive audiences at the Visual Education Program at Houston encouraged the exhibitors to redouble their efforts to bring the latest advances to you this year in Boston.

Plan to attend the American Dental Association Meeting in Boston, Aug. 24 to 28, 1942, and be sure to save plenty of time for the Scientific and Health Exhibits in Mechanics Hall.

LEO F. MARRÉ, Chairman
Scientific and Health Exhibits Committee
Missouri Theatre Building
St. Louis, Mo.

Notes of Interest

Dr. George B. Broadhurst announces the removal of his office from 401-402 University Club Building to 301-304 University Club Building, Grand and Washington, St. Louis, Mo. Practice limited to exodontia and oral surgery.

Dr. Harvey A. Stryker, 450 Sutter St., San Francisco, Calif., announces the opening of an additional office in the Medical Building, 1904 Franklin St., Oakland, Calif. Practice limited to orthodontics.

Dr. Leuman M. Waugh and Dr. Donald B. Waugh are now located at 931 Fifth Avenue, New York City. Practice limited to orthodontics.

OFFICERS OF ORTHODONTIC SOCIETIES*

American Association of Orthodontists

President, J. A. Burrill _ _ _ _ _ _ _ _ _ _ _ 25 East Washington St., Chicago, Ill.
Secretary-Treasurer, Max E. Ernst _ _ _ 1250 Lowry Medical Arts Bldg., St. Paul, Minn.
Public Relations Bureau Director, Dwight Anderson
_ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ _ 292 Madison Ave., New York, N. Y.

Central Association of Orthodontists

President, Harold J. Noyes _ _ _ _ _ _ _ _ _ 55 E. Washington St., Chicago, Ill.
Secretary-Treasurer, L. B. Higley _ _ _ _ _ _ _ 705 Summit Ave., Iowa City, Iowa

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Secretary-Treasurer, Curtis L. Benight _ _ _ _ _ _ 1001 Republic Bldg., Denver, Colo.

Southern Society of Orthodontists

President, W. P. Wood, Jr. _ _ _ _ _ _ _ _ _ _ 442 W. Lafayette St., Tampa, Fla.
Secretary-Treasurer, E. C. Lunsford _ _ _ _ _ _ 2742 Biscayne Blvd., Miami, Fla.

Southwestern Society of Orthodontists

President, E. Forris Woodring _ _ _ _ _ _ _ _ _ Medical Arts Bldg., Tulsa, Okla.
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American Board of Orthodontics

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William E. Flesher _ _ _ _ 806 Medical Arts Bldg., Oklahoma City, Okla.
James D. McCoy _ _ _ _ _ _ _ 3839 Wilshire Blvd., Los Angeles, Calif.
Joseph D. Eby _ _ _ _ _ _ _ _ _ 121 E. 60th St., New York, N. Y.

Harvard Society of Orthodontists

President, Harold J. Nice _ _ _ _ _ _ _ _ _ 475 Commonwealth Ave., Boston, Mass.
Secretary-Treasurer, Edward I. Silver _ _ _ _ _ _ _ 80 Boylston St., Boston, Mass.

Washington-Baltimore Society of Orthodontists

President, Paul W. Hoffman _ _ _ _ _ _ _ 1835 Eye St., N. W., Washington, D. C.
Secretary-Treasurer, Stephen C. Hopkins _ _ _ _ _ 1726 Eye St., Washington, D. C.

Foreign Societies†

British Society for the Study of Orthodontics

President, S. A. Riddett
Secretary, R. Cutler
Treasurer, Harold Chapman

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*The Journal will make changes or additions to the above list when notified by the secretary-treasurer of the various societies. In the event societies desire more complete publication of the names of officers, this will be done upon receipt of the names from the secretary-treasurer.

†The Journal will publish the names of the president and secretary-treasurer of foreign orthodontic societies if the information is sent direct to the editor, 3022 Forsythe, St. Louis, Mo., U. S. A.